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The Importance of Carbohydrate Timing during High-intensity Training while Consuming a Low Carbohydrate Diet

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The effects of low carbohydrate (CHO), high fat (LCHF) diets on adaptations to high-intensity exercise have recently gained interest. Consuming a LCHF may potentially decrease the ability to use CHO during exercise and impair high-intensity exercise adaptations. Therefore, the purpose of this investigation was to examine the importance of CHO timing while consuming a LCHF diet and completing a high-intensity exercise program. 18 resistance trained males were randomized into 2 treatment groups. Both groups completed 6 weeks of a high-intensity exercise training program with the first 2 weeks serving as familiarization to resistance training (RT) 3 days per week and completing one high-intensity interval training (HIIT) session. During the final 4 weeks, participants trained 5 days per weeks, 3 days of RT and 2 days of HIIT (repeated 30 s all out sprints). All participants consumed a LCHF diet (~25%, ~25%, and ~50% of daily kilocalorie intake coming from CHO, protein, and fat). The supplemented (SUPP) group (n=9) consumed 30 g of CHO during exercise and 40 g of CHO immediately after each exercise session. The remainder of the SUPP groups daily CHO intake came outside of training. The non-supplemented (NONSUPP) group (n=9) consumed an artificially

flavored placebo during exercise. The NONSUPP group had the same daily CHO intake as the SUPP group, with the only difference being CHO timing. Dependent variables measured pre-and post-training included back squat and bench press one-repetition maximums, peak oxygen consumption ($\dot{V}O_{2\ peak}$), power output (Wingate test), body composition, fasted glucose, insulin, and testosterone, and gastrointestinal distress (GID) during exercise. Both groups significantly improved back squat and bench press strength, biceps thickness, absolute and relative $\dot{V}O_{2\ peak}$, and power output. Respiratory exchange ratio was significantly lower and time to exhaustion significantly increased during the post $\dot{V}O_{2\ peak}$ test. However, there were no changes in resting glucose, insulin, and testosterone or body fat. RT and HIIT caused significant increases in GID, independent of beverage content, with no differences between training. Our results suggest that CHO timing has no impact on adaptations to exercise training, but favorable training adaptations can be made while consuming a LCHF diet.

DEDICATION

This dissertation is dedicated to my family for providing support throughout my academic journey. My parents Mike and Maureen have always provided encouragement and been there when I needed support. I would also like to thank my sisters Alyssa and Mariah who were supportive of my decision to achieve a doctoral education. The opportunity to continue my education at Mississippi State University would not have been possible without the opportunities provided by my extended family. They provided me with a place to live and employment while searching for my next career opportunity. Overall, I am very thankful for everyone who has supported me along this seemingly never-ending journey.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

High-intensity exercise provides a unique and demanding metabolic stress on the human body. As exercise intensity increases, carbohydrate (CHO) metabolism increases and lipid metabolism decreases (Romijn et al., 1993; van Loon, Greenhaff, Constantin-Teodosiu, Saris, & Wagenmakers, 2001). In comparison to total energy availability from lipids, humans store a significantly lower amount of CHO (Kenney, Wilmore, & Costill, 2015). Therefore, an abundance of research has been conducted to determine the effects of exogenous CHO supplementation on exercise performance. Currently, it is well accepted that CHO supplementation can improve endurance performance lasting greater than one hour (Maughan, Bethell, & Leiper, 1996; Neufer et al., 1987; Smith et al., 2013; Smith et al., 2010). However, there is a paucity of research to support the efficacy of CHO supplementation during exercise lasting less than one hour, specifically resistance training (RT) and high-intensity interval training (HIIT).

During a bout of RT, phosphocreatine and CHO play important roles in providing energy. Over the course of a RT session muscle glycogen depletion is accelerated following numerous RT protocols (Haff et al., 2000; Koopman et al., 2006; Macdougall et al., 1999; Pascoe, Costill, Fink, Robergs, & Zachwieja, 1993; Robergs et al., 1991; Tesch, Colliander, & Kaiser, 1986; Tesch, Ploutz-Snyder, Yström, Castro, & Dudley, 1998). During HIIT when intervals are moderately short, (i.e., 30 seconds) and maximal

effort is given, the metabolic demands are similar to RT (Cochran et al., 2014; Freese, Gist, & Cureton, 2013). Exogenous CHO administered before and/or during RT has been shown to improve RT performance (Haff et al., 2001; Haff et al., 1999; Krings et al., 2016; Lambert, Flynn, Boone, Michaud, & Rodriguez-Zayas, 1991; Oliver et al., 2016) and different variations of HIIT (Krings, Peterson, Shepherd, McAllister, & Smith, 2017; Lee et al., 2014; Pomportes, Brisswalter, Hays, & Davranche, 2016), but there is a large amount of evidence reporting conflicting results (Conley et al., 1995; Dalton, Rankin, Sebolt, & Gwazdauskas, 1999; Fairchild, Dillon, Curtis, & Dempsey, 2016; Haff et al., 2000; Khorshidi-Hosseini & Nakhostin-Roohi, 2013; Kulik et al., 2008; Raposo, 2011; Smith et al., 2017b; Vincent, Clarkson, Freedson, & DeCheke, 1993). CHO supplementation during acute bouts of RT and HIIT may decrease fatigue and improve performance; however, there is minimal research examining the efficacy of CHO supplementation during chronic RT programs (Bird, Tarpenning, & Marino, 2006b; Tarpenning, Wiswell, Hawkins, & Marcell, 2001).

Although CHO are one of the most important macronutrients in both daily nutritional and exercise needs, there has been a recent trend towards promoting lower CHO diets. Low CHO diets (LCD) are usually accomplished by reducing CHO intake and increasing dietary fat intake (LCHF). LCHF diets may potentially improve markers of health (Noakes & Windt, 2017), improve body composition (Volek, Quann, & Forsythe, 2010), provide favorable expression of energy sensing cellular pathways (Draznin, Wang, Adochio, Leitner, & Cornier, 2012; Waldman, Krings, Smith, & McAllister, 2018) or may provide favorable adaptations for endurance athletes (McSwiney et al., 2017; Volek et al., 2016). However, reductions in CHO intake have

been shown to reduce CHO metabolism evident by reductions in pyruvate dehydrogenase and glycogenolysis during submaximal and supramaximal exercise (Stellingwerff et al., 2006). Reductions in CHO metabolism could be detrimental to an individual training to increase high-intensity exercise performance.

There is a limited amount of research examining LCHF diets with high-intensity exercise performance. The current literature suggests that LCHF diets, specifically ketogenic diets do not decrease muscular strength (Kephart et al., 2018; Paoli et al., 2012; Wilson et al., 2017), but when paired with a periodized RT program can improve strength (Wilson et al., 2017). Furthermore, ketogenic diets can promote favorable hormonal adaptations (Wilson et al., 2017). However, ketogenic diets may not be practical for everyone due do strict CHO intake guidelines (<50 g/day). Recently, our lab found that resistance trained males can receive favorable adaptations to a more moderate LCHF diet (~50% fat, ~25% CHO, ~25% protein), including increases in fat oxidation during exercise and reductions in resting insulin (Waldman et al., 2017). Daily CHO intakes of less than 2 $g \cdot kg^{-1} \cdot day^{-1}$ have also been shown to have no detrimental effects on RT performance (Dipla et al., 2008; Mitchell & DiLauro, 1997; Sawyer et al., 2013; Symons & Jacobs, 1989). Although LCHF diets may maintain previously attained RT adaptations, there is minimal research examining LCHF diets during periodized training programs.

Due to the importance of CHO in fueling high-intensity exercise, CHO timing has long been thought to potentially optimize performance and metabolic adaptations to high-intensity exercise training. Specifically, consuming CHO during and after exercise may maintain/increase exogenous CHO metabolism (Cox et al., 2010), attenuate muscle glycogen depletion (Haff et al., 2000), promote a positive muscle protein balance, by

increasing anabolic hormone levels and decreasing muscle protein degradation (Borsheim et al., 2004; Roy, Fowles, Hill, & Tarnopolsky, 2000; Roy, Tarnopolsky, MacDougall, Fowles, & Yarasheski, 1997), and replenish muscle glycogen stores after training (Pascoe et al., 1993; Roy & Tarnopolsky, 1998; Tarnopolsky et al., 1997). Furthermore, the timing of CHO intake around training may become more important when consuming a LCHF diet. Therefore, the purpose of this investigation is to examine the effects of timing of CHO intake while completing a high-intensity training program and consuming a LCHF. The findings from this investigation can provide valuable dietary and fueling strategies to anaerobically trained individuals interested in or currently consuming a LCHF.

CHAPTER II

LITERATURE REVIEW

Resistance Training

Physical activity in the form of RT can provide beneficial adaptations to individuals seeking to increase muscular power, strength, hypertrophy, and endurance. Depending upon the goal of the individual, favorable adaptations can increase performance not only during RT sessions, but also outside of the RT facility. This review will focus on the practices to optimize RT adaptations and how the human body physiologically adapts.

There are several adaptations to RT that lead to positive benefits across many populations. Typical adaptations to RT include increases in muscle tissue, due to muscular hypertrophy. Physiological adaptations improving performance include: neurological adaptations (to both the central and peripheral nervous systems), structural muscle changes, enzymatic activity, connective tissue adaptations, and hormonal responses (Haff & Triplett, 2015). Acute RT leads to conformational changes within skeletal muscle (Gibala et al., 2000; Gibala, MacDougall, Tarnopolsky, Stauber, & Elorriaga, 1995). However, it is well accepted improvements in performance due to skeletal muscle structure changes require at minimum 3 to 4 weeks (Abe, DeHoyos, Pollock, & Garzarella, 2000; Seynnes, de Boer, & Narici, 2007; Stock et al., 2016), but are most commonly seen at 6 weeks and beyond (Ikai & Fukunaga, 1970). Factors such

as periodization and acute training variables also play a role in determining the extent of adaptations.

Periodization

Well-designed RT programs typically utilize a periodized approach. Periodization refers to a systematic training program that manipulates training variables to target specific adaptations while providing adequate overload and recovery. Within a systematic training cycle, variables such as volume, intensity, mode of exercise, and rest periods play an important role in attaining adaptations. Linear, reverse linear, nonlinear undulating, and nonlinear flexible undulating periodization schemes all target specific training goals (i.e. hypertrophy, strength), but manipulate intensity and volume differently (Haff & Triplett, 2015). Linear periodization typically starts with higher volume/lower intensity (training load) at the beginning of a training cycle and increases intensity while decreasing volume over the course of a program. Reverse linear provides a direct opposite training program by starting with higher intensity/lower volume and increasing volume and decreasing intensity over time (Rhea et al., 2003). However, reverse linear periodization is generally targeted towards muscular endurance goals and linear periodization targets hypertrophy, muscular strength, and muscular power. Research suggests both models are effective in improving maximal muscular strength, but linear periodization is superior in performance within a 4 to 14 repetition maximum range (Prestes, De Lima, Frollini, Donatto, & Conte, 2009). Furthermore, nonlinear and nonlinear flexible periodization schemes target more frequent changes in volume and intensity and provide similar strength adaptations to traditional linear periodization (Buford, Rossi, Smith, & Warren, 2007). Nonlinear flexible periodization can also be

referred to as undulating periodization. Depending upon the level of training background and genetic ceiling of an individual, it is expected that a high level of muscular adaptations can be obtained, but not without a well-periodized RT program.

Adaptations to Resistance Training

Neuromuscular adaptations. Following a periodized RT program, two of the primary adaptations are neuromuscular and structural changes. Neuromuscular changes typically occur before structural changes and are characterized by greater neuromuscular activation, evident be changes in electromyography activity (Baroni et al., 2013; Hakkinen, Alen, & Komi, 1985; Hakkinen & Komi, 1983; Higbie, Cureton, Warren, & Prior, 1996). Factors leading to greater neuromuscular activation following RT are increased motor unit synchronization (Semmler, 2002; Semmler, Sale, Meyer, & Nordstrom, 2004), decreased neuromuscular inhibition (Aagaard et al., 2000), and increased motor unit firing rate (Aagaard, Simonsen, Andersen, Magnusson, & Dyhre-Poulsen, 2002). Baroni et al. (2013) examined rate of neurological adaptation during a 12-week knee extensor eccentric training program and observed significant increases in neuromuscular activation following 4 weeks of RT. Other investigations have observed similar time to neuromuscular adaptations following RT (Bemben & Murphy, 2001; Del Balso & Cafarelli, 2007; Tillin & Folland, 2014).

Skeletal muscle adaptations. Muscle fiber hypertrophy results from a combination of more myofibrils, sarcoplasm, actin and myosin, connective tissue, or a combination of each of these factors (Kenney et al., 2015). More specifically, myofilaments and sarcomeres typically increase number in parallel fashion (Paul &

Rosenthal, 2002). Muscle fiber hypertrophy can be measured by using ultrasound, magnetic resonance imaging (MRI), and muscle biopsy techniques. Several investigations have found significant increases in muscle cross sectional area (CSA) following various RT programs, potentially indicating muscle fiber hypertrophy utilizing the ultrasound technique (Abe et al., 2000; Blazevich, Cannavan, Coleman, & Horne, 2007; Blazevich, Gill, Bronks, & Newton, 2003; Starkey et al., 1996). Due to the cost of MRI machines and invasive nature of muscle biopsies (Bergstrom & Hultman, 1966), the ultrasound technique provides a cost effective noninvasive method to quantify muscle thickness.

For muscle hypertrophy to occur a series of factors including: satellite cells, myogenic pathways, and hormones play a role in the adaptive process (Schoenfeld, 2010). Satellite cells are stem cells located on the plasma membrane and basal lamina of a muscle fiber and play an important role in growth and generation of muscle (Bazgir, Fathi, Rezazadeh Valojerdi, Mozdziak, & Asgari, 2017). When stimulated, satellite cells give a nuclei to the muscle fiber (Moss & Leblond, 1971), leading to the ability to generate new tissue. Myogenic pathways refer to molecular signaling mechanisms responsible for hypertrophy. Mammalian target of rapamycin complex 1 (mTORC1), calcium-dependent pathways, and mitogen-activated protein-kinase pathways are believed to be the signaling pathways (Schoenfeld, 2010) responsible for muscle protein synthesis. mTORC1 has been termed the “master” regulator for protein synthesis and is promoted by RT and inhibited when in a state of catabolism. In brief, following a muscle contraction (i.e. mechanical stress) the protein kinase Akt is activated and its downstream target is mTORC1 via phosphorylation of ribosomal kinases (Huang & Manning, 2009).

Several other factors, such as hormones, nutrition, and interaction with other signaling pathways, and their influence on mTORC1 will be discussed in detail later in this review.

Mitogen-activated protein kinases (MAPK) play a role in several physiological processes, most notably gene expression. Following a bout of RT in young men, significant increases in extracellular signal regulated kinase (ERK) ½, p38 MAPK, and c-Jun N-terminal and kinase/stress activated protein kinases (JNK/SAPK) activate gene expressions to elicit muscle fiber development (Williamson, Gallagher, Harber, Hollon, & Trappe, 2003). ERK, p38 MAPK, and JNK/SAPK all play an important role in a signaling cascade responsible for skeletal muscle hypertrophy.

Finally, another class of muscle hypertrophy regulators are calcium-dependent pathways. Calcineurin is generally recognized as the most important regulator responding to changes in calcium levels. Following an influx of cellular calcium, calcineurin is activated and subsequently dephosphorylates transcription factors in the cytoplasm (Hogan, Chen, Nardone, & Rao, 2003) and is thought to control fiber type shifting following RT (Naya et al., 2000). Although myogenic pathways play an important role in cellular adaptations they are often difficult to measure and in turn, researcher's exam hormones due to their regulation of similar pathways.

Hormones play an important role in both the acute and chronic responses to RT. Elevations in anabolic hormones can lead to positive responses within the body, whereas elevations in catabolic hormones potentially mitigate anabolic responses and inhibit downstream myogenic pathway stimulation. However, a balance of both catabolic and anabolic hormone concentrations is necessary for maintenance of substrate availability during exercise and promotion of positive adaptations. Before, during, and following a

single bout of a one-hour resistance exercise session, McMillan et al. (1993) examined hormonal responses, substrate usage, and substrate availability. Their research concluded human growth hormone, catecholamines (epinephrine and norepinephrine), and cortisol play an important role in maintaining substrate availability during exercise. Furthermore, the importance of the primary anabolic hormones of testosterone, human growth hormone, insulin, and insulin-like growth factor, play a significant role in the adaptive process following resistance exercise (Kraemer & Ratamess, 2005).

Resting testosterone levels from chronic RT have been suggested to be a marker of RT stimulus, due to testosterones capability to stimulate protein synthesis and inhibit protein degradation (Vingren et al., 2010). Increases in resting testosterone have been observed following RT programs lasting 4 weeks (Staron et al., 1994), 6-8 weeks (Kraemer et al., 1998a), 6-10 weeks (Kraemer et al., 1999), 12 weeks (Moradi, 2015), and 14 weeks (Ahtiainen, Pakarinen, Alen, Kraemer, & Hakkinen, 2003). However, RT variables including rest period length, exercise, order of exercise, intensity, number of sets, and total volume effects both acute and resting testosterone levels (Vingren et al., 2010). Ahtiainen et al. (2003) observed increases in testosterone following two 7-week high-volume RT cycles, but then reduced training volume during a final 7-week cycle leading to a decrease in resting testosterone. Decreases in training volume have also been observed to reduce resting testosterone levels in elite weight lifters (Hakkinen, Pakarinen, Alen, Kauhanen, & Komi, 1987, 1988). High levels of resting testosterone may be an indicator of anabolic status but are influenced by a multitude of factors.

Training status can also play a large role in hormonal responses to exercise. When comparing anabolic and catabolic hormone response following a bout of RT, experienced

weight lifters exhibit a significantly lower anabolic hormone response (Cadore et al., 2008). This suggests that an adaptation to RT initially may be increases in anabolic hormone responses, but as training age increases, anabolic hormone response becomes less important. More recently, it has been observed that acute hormone responses, both anabolic and catabolic hormones, do not correlate to chronic muscle strength and hypertrophy adaptations (Morton et al., 2016). Other factors, such protein and CHO, have also been suggested and shown to enhance the anabolic hormone response to RT (Kraemer, Volek, Bush, Putukian, & Sebastianelli, 1998b). Hormonal responses play an integral role in the adaptive process following RT, however, recent evidence suggests that chronic resting hormone concentrations may be a better marker of adaptation than acute responses. Nonetheless, RT variables, nutrition, and training status play an important process in hormone status through a RT program.

It should be mentioned; net protein balance needs to be positive for any type of skeletal muscle fiber hypertrophy to occur. Positive protein balance is controlled by the sum of muscle protein breakdown and muscle protein synthesis (Phillips, 2014). Resistance exercise creates protein breakdown and synthesis in the muscle (Biolo, Maggi, Williams, Tipton, & Wolfe, 1995), but macronutrients, specifically protein, is needed to further augment synthesis and promote a positive protein balance (Tipton et al., 2004). Therefore, dietary intake and nutrient timing play an important role in regulating physiological adaptations to RT.

Substrate Utilization During Resistance Training

RT provides a unique metabolic demand requiring adequate energy from the phosphocreatine system, glycolytic, and oxidative systems for energy production and

recovery. During high-intensity bouts of RT, energy production predominately comes from glycolytic metabolism. During a 30 min bout of lower body RT, Tesch et al. (1986) observed significant reductions in ATP, phosphocreatine, and glycogen, with significant increases in glucose and glucose-6-phosphate concentrations. Additionally, reductions in muscle glycogen have been observed during upper body RT (Macdougall et al., 1999), lower body dynamic training (Pascoe et al., 1993; Robergs et al., 1991; Tesch et al., 1986; Tesch et al., 1998), and lower body isokinetic training (Haff et al., 2000). CHO metabolism rate has also been shown to be different between muscle groups, as the leg press exercise significantly increases CHO metabolism compared to the chest fly exercise (Farinatti, Castinheiras Neto, & Amorim, 2016). Regardless of the muscle mass activated, CHO metabolism increases from the beginning of exercise to the final set, highlighting the importance of CHO in RT (Farinatti et al., 2016).

A positive adaptation to endurance training is increasing lipid metabolism and relying less on CHO metabolism while still increasing exercise performance. For RT individual reducing the reliance on CHO without hindering performance would be a favorable adaptation. Dietary factors potentially effecting metabolism during RT will be addressed. Specifically, the need of CHO during RT while significantly reducing daily CHO intake and increasing daily fat intake.

High Intensity Interval Training

HIIT is a broad term referring to short bouts of high-intensity repeated exercise. This type of training is time efficient and elicits similar adaptations to traditional long, slow, submaximal aerobic exercise (Burgomaster et al., 2008; Cochran et al., 2014; Gillen et al., 2016; Rakobowchuk et al., 2008). One of the most common forms of HIIT

requires 4-6 repeated 30-s all-out effort bouts of exercise separated by four min of active recovery (Gibala, Little, Macdonald, & Hawley, 2012). Recently an investigation reported improved aerobic fitness similar to that of traditional aerobic training in sedentary men, but at a five-fold lower time commitment (Gillen et al., 2016). Due to time constraints in daily schedules, individuals can practice some form of HIIT to elicit positive physiological adaptations in a time effective manner.

Adaptations to High Intensity Interval Training

A series of investigations performed at McMaster University have repeatedly examined the physiological and performance adaptations to HIIT incorporating repeated bouts of 30-s all out sprints (Burgomaster et al., 2007; Burgomaster, Heigenhauser, & Gibala, 2006; Burgomaster et al., 2008; Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Cochran et al., 2014; Gibala et al., 2006; Hazell, Macpherson, Gravelle, & Lemon, 2010; MacDougall et al., 1998; Rakobowchuk et al., 2008). This research has led to a large cohort of investigations. Significant adaptations to HIIT have been observed in as little as six training sessions (Astorino, Allen, Roberson, & Jurancich, 2012; Burgomaster et al., 2006; Burgomaster et al., 2005; Gibala et al., 2006). Physiological adaptations to HIIT include increases in: oxidative enzyme capacity (Burgomaster et al., 2006; Burgomaster et al., 2008; Burgomaster et al., 2005; Gibala et al., 2006; MacDougall et al., 1998), arterial distensibility and endothelial function (Rakobowchuk et al., 2008), stroke volume (Trilk, Singhal, Bigelman, & Cureton, 2011), muscle capillarization (Cocks et al., 2013), mean power output (Whyte, Gill, & Cathcart, 2010), and peak power output (Astorino et al., 2012). Participating in HIIT offers an important

training stimulus eliciting favorable adaptations to many different populations (i.e. sedentary, obese, active) in a time efficient manner.

One of the most important adaptations to HIIT, often not attained with long slow continuous aerobic exercise, is an increase in anaerobic power output. Power output is an important performance variable for individuals competing in anaerobic activities and RT. Improvements in relative peak and mean power output (Astorino et al., 2012; Hazell et al., 2010) as well as absolute peak and mean power output (Burgomaster et al., 2006) have been observed in recreationally active men and women following 2 weeks of HIIT. During a 6-week investigation, Burgomaster et al. (2008) compared HIIT and long, slow, continuous, aerobic training observing significant increases in peak power with both groups (17% and 7%, respectively), but no differences between groups, and mean power increased in the HIIT group only. Using HIIT compared to traditional aerobic exercise may be an important option for athletes wanting to improve aerobic capacity while maintaining and/or increasing anaerobic power output.

Substrate Utilization During High Intensity Interval Training

HIIT can encompass a wide range of short duration exercise bouts (~5 s to 4 min), however this review will address metabolism for 30 s sprints. HIIT produces similar metabolic demands as RT, however HIIT relies increasingly more on aerobic metabolism. During a 30-s, maximal sprint on a treadmill, there are significantly greater reductions in phosphocreatine and muscle glycogen of type II fibers compared to type I fibers (Greenhaff et al., 1994). Similar responses have been observed during cycling exercise (Casey, Constantin-Teodosiu, Howell, Hultman, & Greenhaff, 1996). Relative energy contribution during a 30-s all out cycling sprint has been identified as 17%, 38%,

and 45% coming from ATP and phosphocreatine, oxidative, and anaerobic glycolysis (Medbo, Gramvik, & Jebens, 1999). When a second 30-s sprint is completed following four minutes of recovery, energy contribution from oxidative metabolism provides ~49% of energy production and anaerobic production (glycolysis) decreases by ~41% (Bogdanis, Nevill, Boobis, & Lakomy, 1996). Completing two more sprints, for a total of four 30-s sprints, results in a plateau in aerobic metabolism as evident by the measurement of relative oxygen consumption (Freese et al., 2013). Due to the dependency of energy production through substrate level CHO phosphorylation and CHO oxidation, HIIT produces a unique metabolic demand and improves both anaerobic and oxidative capacity.

Concurrent Training

Concurrent training is described as completing both aerobic and RT in the same periodized training program. Many athletes and active individuals train concurrently to meet minimal physical activity guidelines. However, this concept has received negative implications due to the proposed interference effect of concurrent training (Wilson et al., 2012). The interference effect was first explored in the 1980's (Hickson, 1980). This investigation examined the effects of training endurance and strength in the same day, training endurance only, and training strength only for a total of 10 weeks. The results suggested the strength only group increased strength greater than the concurrent training group (Hickson, 1980). Similar results were observed following a 12-week training program, with a concurrent training group exhibiting significantly lower 1RM unilateral knee extension strength compared to a strength only group (Bell, Syrotuik, Martin, Burnham, & Quinney, 2000). Concurrent training has also been shown to negatively

affect muscle power adaptations following 22 weeks of training (Hakkinen et al., 2003). However, there has been contention to the negative implications of concurrent training, as 10 weeks (McCarthy, Pozniak, & Agre, 2002) and 22 weeks (Sale, MacDougall, Jacobs, & Garner, 1990) of strength and concurrent training produced similar muscular hypertrophy gains of the quadriceps. Due to disparity of research findings, it is difficult to fully elicit a strong conclusion on the proposed interference effect of concurrent training. It is also important to note, endurance adaptations are improved following concurrent training to the same extent as endurance only training groups, leading to a large amount of literature emphasizing strength adaptations.

Recent advances in molecular signaling detection surrounding endurance and strength training have allowed researchers to better understand implications of the interference effect. Interactions between the signaling cascades promoting muscle hypertrophy and mitochondrial biogenesis have been suggested to negatively impact adaptations to muscular strength, hypertrophy, and power (Baar, 2014; Fyfe, Bishop, & Stepto, 2014). In a recent meta-analysis, Wilson et al. (2012) observed the factors of endurance modality and training volume significantly affect concurrent training adaptations. Decreasing overall endurance training volume by completing HIIT (Balabinis, Psarakis, Moukas, Vassiliou, & Behrakis, 2003) instead of traditional endurance training and completing cycling instead of running (Wilson et al., 2012) are two possible options to minimize the interference effect. Another important factor in concurrent training is the time between training sessions. In a large study using amateur rugby players, participants underwent 7 weeks of concurrent training in one of three groups: strength training and endurance training completed back-to-back, separated by

six hours, and separated by 24 hours (Robineau, Babault, Piscione, Lacome, & Bigard, 2016). The authors concluded concurrently training with 6 hours between sessions is the minimum amount of time needed to minimize the interference effect, but 24 hours is optimal for the greatest neuromuscular and strength adaptations (Robineau et al., 2016). Completing high-intensity cycling on a different day from RT may be the most ideal training schedule to promote positive muscular adaptations to concurrent training.

The importance of nutrition has also been suggested as a potential mechanism to mitigate the interference effect of concurrent training (Perez-Schindler, Hamilton, Moore, Baar, & Philp, 2015). Due to the influence of nutrition, specifically proteins and CHO, on molecular signaling pathways, a multistep approach has been suggested: begin the day with an endurance session and train resistance exercise in the afternoon, include protein ingestion surrounding all training sessions and ingest CHO around the resistance exercise (Perez-Schindler et al., 2015). Nutrition may often be overlooked in the aspect of concurrent training, but the importance of nutrition supporting both RT and aerobic exercise independently, suggests that nutritional intake and timing strategies play an important role in maximizing adaptations to concurrent training.

Carbohydrates

Structure/Types

CHO are the bodies main fuel source during exercise and typically make up a large portion of daily macronutrient intake. All CHO have the same basic structure, which includes: carbon, oxygen, and hydrogen, and are formed into two major classes, simple and complex. Depending upon the number of carbons, CHO can be labeled as trioses, tetroses, pentoses, hexoses, and heptoses (Gropper & Smith, 2012). CHO appear

in two configurations of L or D. Within the human body, only D configuration can be broken down and metabolized. Furthermore, CHO structure is either in the α or β configuration, determining the bond between the carbon-oxygen at the anomeric carbon (Gropper & Smith, 2012).

Simple CHO are composed of either monosaccharides or disaccharides. The three monosaccharides are glucose, fructose, and galactose. Disaccharides are composed of two monosaccharides. Glucose is the most important monosaccharide as it is part of the disaccharides humans use. During exercise, the type of CHO ingested is important as multiple transportable CHO can maximize exogenous oxidation compared to single CHO (Jentjens, Achten, & Jeukendrup, 2004; Jentjens & Jeukendrup, 2005; Jentjens et al., 2005; Jentjens et al., 2006; Jentjens, Venables, & Jeukendrup, 2004). This fueling strategy may be extremely important during ultra-endurance activities (Burke, Hawley, Wong, & Jeukendrup, 2011).

Complex CHO are composed of polysaccharides or oligosaccharides. Oligosaccharides are a short chains of monosaccharides, usually three to ten monosaccharides (Gropper & Smith, 2012). Due to the inability of digestive enzymes within the gastrointestinal system to digest oligosaccharides, they are broken down by gut bacteria. In comparison, polysaccharides are long chains of monosaccharides and are more common than oligosaccharides. Two of the most common polysaccharides are glycogen and starch. Amylopectin and amylose are the two forms of starch, consisting of 80 to 85%, and 15 to 20% of starch content in food, respectively (Gropper & Smith, 2012). All polysaccharides contain long chains of glucose, but glycogen can contain

chains of 100-1000's of glucose molecules. Glycogen supports energy availability by storing glucose within the liver and muscle.

Digestion

The overall goal of CHO digestion is to break down multiple chained CHO into monosaccharides. Polysaccharide digestion begins in mouth with salivary α - amylase and continues in the gut and small intestine. Disaccharides are mainly digested within the small intestines brush border of the microvilli (Gropper & Smith, 2012). Depending upon the disaccharide, sucrase (sucrose), maltase (maltose), lactase (lactose), trehalase (trehalose) catalyze further breakdown into their respective monosaccharides.

Absorption

Facilitation of CHO cellular uptake is dependent on the type of CHO. Following a meal rich in CHO and subsequent digestion, CHO are absorbed into the blood stream. Glucose and galactose are absorbed by active and/or facilitated diffusion. Active diffusion occurs with the cost of energy, via sodium-potassium ATPase pump. The sodium glucose transporter 1 (SGLT-1) is then brought to the microvilli receptor and transports both monosaccharides into the intestinal lumen. From there, GLUT-2 transporters facilitate the transport to the blood stream. However, when large amounts of glucose and galactose are ingested, transport to the blood stream can occur without the cost of energy by GLUT-2. Fructose requires transport via GLUT-5 into the intestinal wall and GLUT-2 is also responsible for fructose transport to the blood stream (Riby, Fujisawa, & Kretchmer, 1993).

Upon entry to the blood stream, monosaccharides travel to the liver. Most fructose and galactose are metabolized immediately by the liver. Once in the blood stream glucose can enter the targeted tissue. However, the facilitation of diffusion is dependent upon the tissue. For example, tissues that require glucose as their predominate energy source, red blood cells and the brain, bring glucose into the cell unregulated by GLUT-1 and GLUT-3 transporters, respectively (Tiidus, Tupling, & Houston, 2012). Uptake of blood glucose into the muscle is mainly regulated by insulin, which is secreted from the β cells of the pancreas. Insulin-dependent glucose uptake via GLUT-4 transport into skeletal muscle accounts for 70-80% of insulin-stimulated glucose uptake (Tiidus et al., 2012). Glucose homeostasis is regulated by the liver and controlled by the following process: initial insulin secretion, stimulation of glucose uptake from the peripheral tissues and splanchnic tissues, and suppression of liver glucose production (DeFronzo & Ferrannini, 1987).

Metabolism

Six carbon glucose molecules are broken down into two 3-carbon phosphates, glyceraldehyde 3-phosphate (G3P) and dihydroxyacetone phosphate (DHAP; Tiidus et al., 2012). This process occurs in glycolysis, the primary pathway of CHO metabolism. In the presence of oxygen, the final product of G3P and DHAP is two three carbon molecules of pyruvate. In contrast, anaerobic metabolism, or fast glycolysis, results in the conversion of pyruvate to lactate. Glycolysis is regulated by several modulators including: levels of adenosine triphosphate (ATP), hydrogen ions, citrate, adenosine diphosphate, adenosine monophosphate, and inorganic phosphate (Tiidus et al., 2012). Other pathways of CHO metabolism include: glycogenolysis (degradation of glycogen to glucose), glycogenesis (synthesis of glucose to glycogen), gluconeogenesis (synthesis of

glucose from non-CHO), Krebs' cycle (oxidation of acetyl-coA to water), and pentose phosphate pathway (synthesis of three to seven carbon sugars and nicotinamide adenine dinucleotide phosphate). During short-duration intense exercise, such as RT, ATP is supplied from ATP-PCr and glycolysis, especially during high volume training.

Nutrient Manipulation

Nutritional supplementation surrounding exercise is implemented with the goals of supporting performance, promoting positive physiological adaptations, and improving recovery. Any type of nutrient supplemented, with the overall goal of enhancing performance, is described as an ergogenic aid (Thein, Thein, & Landry, 1995). Beneficial effects from ergogenic aids have been observed with multiple supplements. Ergogenic aids that are well supported in sports nutrition include: CHO (Smith et al., 2013; Smith et al., 2010), protein (Reidy et al., 2013; Tipton et al., 2004), amino acids (Karlsson et al., 2004; Tipton, Ferrando, Phillips, Doyle, & Wolfe, 1999), caffeine (Bruce et al., 2000; Souissi et al., 2012), and creatine (Bemben, Bemben, Loftiss, & Knehans, 2001; Pearson, Hamby, Russel, & Harris, 1999). One of the most extensively studied supplements is CHO. Although many individuals obtain CHO from dietary intake, exogenous CHO supplementation may promote beneficial results during anaerobic (Baker, Rollo, Stein, & Jeukendrup, 2015) and aerobic (Stellingwerff & Cox, 2014) exercise.

Carbohydrate Manipulation

Recently, there have been advances in sports nutrition suggesting the importance of nutrient manipulation surrounding aerobic training (Hawley & Burke, 2010). Marquet et al. (2016a) examined the effects of CHO periodization during a short-term 3-week

endurance training intervention and observed improvements in submaximal and supramaximal performance after providing low CHO availability (commencing training with low glycogen status) during low intensity long duration training and high CHO availability during high intensity training. In a follow-up Marquet et al. (2016b) observed similar submaximal and supramaximal performance adaptations, but only after 1-week of manipulating CHO availability. Training with low CHO availability has been shown to upregulate several signalers that act on mitochondrial biogenesis, *p*53 (Bartlett et al., 2013) and 5'AMP-activated protein kinase (AMPK; Impey et al., 2016; Wojtaszewski et al., 2003). Furthermore, endurance training with high CHO availability may also increase glucose oxidation during exercise (Cox et al., 2010). Athletes focusing on endurance sports may benefit from this type of nutritional manipulation.

Manipulating CHO availability has also been employed during periods of intensified HIIT. Cochran et al. (2015) examined the short-term (2 weeks) effects of training twice daily with HIIT and CHO availability. The authors found significant increases in time trial performance in the group who commenced their second daily training session with only 17 g of CHO between exercise bouts, but no differences in metabolic adaptations between a group receiving 195 g of CHO (Cochran et al., 2015). HIIT with low CHO availability may improve short term adaptations, but long-term benefits are unknown.

Training with low glycogen status may be beneficial for aerobic adaptations, but it may be argued that to maximize anaerobic adaptations, nutritional support, specifically CHO, surrounding training is important. Muscle protein synthesis is controlled by mTORC1. Currently, research suggests mTORC1 is upregulated by several different

factors, including: insulin, resistance exercise, and leucine (Drummond, Dreyer, Fry, Glynn, & Rasmussen, 2009). Conversely, factors that upregulate mitochondrial biogenesis (an adaptation to aerobic training), are proposed to negatively impact mTORC1 and subsequent protein synthesis. Both amino acids and CHO have been shown to upregulate mTORC1 independent of exercise (Fujita et al., 2007), in combination with RT (Dreyer et al., 2008) and during high-intensity cycling (Ferguson-Stegall et al., 2011; Ivy, Ding, Hwang, Cialdella-Kam, & Morrison, 2008). When assessing protein synthesis during exercise and periods of low CHO availability, there may be a negative impact on protein balance due to reductions in protein synthesis (Howarth et al., 2010). It is also important to note that even in the presence of leucine supplementation with low CHO availability, reductions in ribosomal protein S6 kinase beta-1 (p70S6K) activity are seen, a protein kinase that activates mTORC1, signifying the importance of muscle glycogen stores in regulating protein synthesis (Impey et al., 2016). Due to the limited evidence surrounding CHO manipulation studies and RT it is difficult to conclude the efficacy of training with low or high CHO availability on RT performance.

Carbohydrate Timing

Unlike CHO manipulation, where dietary CHO is specifically adjusted to train in a state of low or high CHO, CHO timing refers to variations in the specific time at which daily CHO is ingested. In one of the few studies examining supplement timing and RT, Cribb and Hayes (2006) examined the effects of ingesting a supplement beverage containing CHO (43 g), protein (40 g), and creatine (7 g). Participants ingested the beverage in the morning and afternoon or immediately before and after RT 4 days per

week for 10 weeks, and the authors observed increases in fat free mass and muscular strength in the before and after training group (Cribb & Hayes, 2006). However, due to the ingestion of creatine, it is difficult to fully conclude the effect CHO had on training adaptations, especially since participants were instructed to maintain their habitual diets. Other investigations have suggested chronic RT while supplementing protein + CHO beverages compared to CHO only before and after RT (Willoughby, Stout, & Wilborn, 2007) as well as other times during the day not around RT (Ballard, Clapper, Specker, Binkley, & Vukovich, 2005) promote more positive adaptations. Based on previous literature, the primary research questions have been around protein supplementation and less with CHO. Therefore, more randomized control investigations are needed to determine the effects of CHO timing surrounding RT when protein is matched between training groups.

Much premise for nutrient timing research surrounding RT is based upon suggestions of taking advantage of the "post-exercise anabolic window". This implies there is a limited amount of time to ingest protein and CHO after exercise to obtain positive adaptations. Some researchers suggest meeting daily macronutrient and total energy needs may support muscle hypertrophy goals the same as nutrient timing (Aragon & Schoenfeld, 2013). However, it may be important for individuals to practice nutrient timing to ensure consumption of protein and CHO around exercise, especially when there is a potential for repeated bouts of exercise in the same day. There is still a limited amount of research to fully elicit a consensus of the importance of nutrient timing, but there may be more of an importance when habitual diet changes.

Low Carbohydrate Diets

The influence of diet on body composition and performance has produced a plethora of scientific research. Of recent, a large majority of research has been focused on LCD. LCD are commonly associated with an increase in dietary protein consumption and/or fat consumption. Anything lower than 45% of total daily caloric intake coming from CHO has been considered a LCD (Aragon et al., 2017). Extreme LCD below 50 grams of CHO per day with a moderate protein intake (1.2-1.5 g/kg/day) and a majority of calories (i.e., >70%) coming from fats is described as a ketogenic diet. Ketogenic diets produce a state of ketosis and the body increases fat metabolism at rest and during exercise (Volek et al., 2016). Ketogenic diets have recently been studied in endurance athletes and found to be inferior to higher CHO diets in elite race walkers (Burke et al., 2017). However, Wilson et al. (2017) found no detrimental effects to performance adaptations or health between ketogenic dieting and traditional western dieting following 8 weeks of periodized RT. Although elite endurance athletes may benefit from high CHO diets, there are mixed opinions on the efficacy of LCD in individuals who predominately resistance train (Escobar, VanDusseldorp, & Kerksick, 2017; Haff & Whitley, 2002). Therefore, this review will focus on LCD and its effects on performance, body composition, insulin, and testosterone.

Current Recommendations

The acceptable macronutrient distribution range for adults is set at 45-65% of total daily caloric intake coming from CHO. Since government mandated nutrition guidelines are geared towards the general population, sports nutrition governing bodies and researchers have set more specific dietary guidelines for athletes (Burke et al., 2011;

Thomas, Erdman, & Burke, 2016a, 2016b). However, current daily CHO intake guidelines for athletes are largely based on endurance exercise (Burke et al., 2011; Jeukendrup, 2011) and applying these CHO guidelines to resistance trained athletes has come under scrutiny (Escobar et al., 2017). For example, the International Society of Sports Nutrition (ISSN) suggests daily CHO intakes of 5-10 g/kg/day (Kreider et al., 2010), but research suggests that lower CHO diets elicit no detrimental effects on RT performance (Dipla et al., 2008; Escobar, Morales, & Vandusseldorp, 2016; Gregory, Hamdan, Torisky, & Akers, 2017; Hatfield et al., 2006; Kephart et al., 2018; Mitchell & DiLauro, 1997; Paoli et al., 2012; Sawyer et al., 2013; Symons & Jacobs, 1989; Van Zant, Conway, & Seale, 2002; Wilson et al., 2017). Due to the differences in metabolic demands of endurance and RT, LCD may be adequate and/or beneficial to promote positive RT adaptations.

Effects of Low Carbohydrate Diets on Performance

Due to the vast array of macronutrient distributions of research studies, research examining the effects of LCD and RT performance is based upon a wide range of CHO intakes: 0.18 g/kg/day (Kephart et al., 2018), 0.32 g/kg/day (Paoli et al., 2012), 0.37 g/kg/day (Mitchell & DiLauro, 1997), 0.39 g/kg/day (Wilson et al., 2017), 0.41 g/kg/day (Sawyer et al., 2013), 0.58 g/kg/day (Gregory et al., 2017), 1.78 g/kg/day (Dipla et al., 2008), 1.8 g/kg/day (Symons & Jacobs, 1989), 3.13 g/kg/day (Escobar et al., 2016), 4.2 g/kg/day (Van Zant et al., 2002), and 4.4 g/kg/day (Hatfield et al., 2006). This range is important because it provides evidence that moderately LCD (Hatfield et al., 2006; Van Zant et al., 2002) and very LCD (Gregory et al., 2017; Kephart et al., 2018; Paoli et al., 2012; Sawyer et al., 2013; Wilson et al., 2017) may not negatively impact performance.

However, it should be noted that differences in study methodologies play an important role in applying dieting strategies to athletes.

Wilson et al. (2017) utilized an 8-week RT program and concluded a ketogenic diet can produce similar strength adaptations as a normal western diet and produce more favorable anabolic hormonal changes. Similarly, Paoli et al. (2012) studied the effects of very LCD for 30 days of RT in male gymnast and found no reduction in performance with squat jumps, jump height, push-ups, and parallel dips compared to the gymnast on their western diet. During a 3-week moderately LCD, Van Zant et al. (2002) found sedentary, aerobic, and resistance trained individuals to have no reductions in performance (maximum bench press, bench press endurance, isokinetic peak torque, and total work) compared to high CHO diet. Recently, ketogenic dieting with CrossFit training has been shown improve performance following 6 weeks (Gregory et al., 2017) and not impact performance following 12 weeks (Kephart et al., 2018). Based upon LCD investigations lasting 3 weeks or longer, it can be hypothesized that RT adaptations and performance may be unaffected by lower daily intakes of CHO.

In a recent position stand on dieting, the ISSN suggested diet studies less than 4 weeks do not provide an adequate time for an individual to adapt to a specific diet and observe diet influenced performance effects (Aragon et al., 2017). Due to the difficulty and cost of assessing long-term dietary interventions, researchers often examine the effects of shorter-duration LCD interventions. One week investigations have observed no differences in peak torque or maximal hand grip strength (Dipla et al., 2008) and maximal bench press strength and upper-body power (Sawyer et al., 2013). Furthermore, shorter duration investigations of four days (Hatfield et al., 2006), three days (Escobar et

al., 2016), and two days (Mitchell & DiLauro, 1997; Symons & Jacobs, 1989) show no decrements in RT performance following a LCD. To date, one study has observed significant reductions in RT performance because of a two-day LCD (1.2 g/kg/day) (Leveritt & Abernethy, 1999). Leveritt and Abernethy (1999) observed significant reductions in squat performance until failure, but not isokinetic strength and the authors alluded to possible reductions in muscle glycogen being the reason for performance detriments. One of the main arguments with LCD is that it puts an individual in a state of low muscle glycogen, which would in turn decrease glycogen availability. However, recent evidence suggests that a long-term adaptation to LCD, specifically ketogenic diets, does not result in reductions in muscle glycogen stores in ultra-endurance athletes (Volek et al., 2016). As discussed further in this review, muscle glycogen is an important fuel source during RT, but may not be the limiting factor of performance, especially based on current scientific literature. Additionally, low glycogen status may not inhibit gene expression and molecular signaling associated with the adaptive response of RT (Camera et al., 2012)

Based on the current evidence, there does not seem to be a detrimental effect of LCD on RT performance. However, due to variations in study populations and diet adaptation periods, more research is needed to establish the efficacy of LCD with RT, specifically dietary interventions lasting 4 weeks or longer.

Effects of Low Carbohydrate Diet on Body Composition

A goal of a LCD may be to improve body composition. Body composition is composed of two components, fat free mass and fat mass, and maintaining/increasing fat free mass (lean body mass) while decreasing fat mass is a common goal amongst RT

individuals. Following a chronic LCD, Paoli et al. (2012) observed significant reductions in body mass and attributed the reductions due to fat mass, but measured body composition via skinfolds and not a "gold standard" body composition assessment (i.e. hydrostatic weighing, dual-energy X-ray absorptiometry). During periods of LCD, as explained above, muscle glycogen stores are expected to decrease. Each molecule of muscle glycogen carries ~3 g of water, so it may be hypothesized water mass is artificially effecting body composition conclusions with LCD research. Similar to Paoli et al. (2012), significant reductions in body mass have been seen during chronic ketogenic diets (Gregory et al., 2017; Kephart et al., 2018; Wilson et al., 2017). Wilson et al. (2017) found that after 8 weeks of RT, the LCD group had significantly greater reductions in fat mass, but from weeks 10-12, CHO were reintroduced into the participants diets and the LCD group showed significant increases in fat free mass (Wilson et al., 2017), suggesting the influence of glycogen on body mass. The conclusions of research investigating LCD may be dependent upon body composition analysis methods.

Effects of a Low Carbohydrate Diet on Resting Insulin

Insulin is polypeptide hormone secreted from the β cells of the pancreas with the primary goal of maintaining glucose homeostasis. Insulin can act as an anabolic hormone due to its ability to bring amino acids and CHO into the muscle to be utilized for fuel and stimulation of protein synthesis. However, elevated resting insulin can lead to deleterious effects such as diabetes, metabolic syndrome, and cardiovascular disease (Grundy et al., 2005). Consuming a LCD may improve homeostatic levels of blood glucose, herein returning resting insulin levels to a healthy range. Previous investigations have observed significant reduction in resting insulin levels with after following a LCD in

unhealthy/sedentary adults (Volek et al., 2009), middle-aged healthy adults (Sharman et al., 2002; Urbain et al., 2017; Volek et al., 2001; Volek et al., 2002), and college-aged resistance trained males (Waldman et al., 2017). During 15 days of consuming a LCD, our lab observed significant reductions in insulin following 5, 10, and 15 days of commencing the diet (Waldman et al., 2017). The results from this study suggests that an already healthy population can significantly reduce resting insulin levels. Resting insulin may be significantly reduced in older and unhealthy populations, but more research with young healthy-populations is needed. Specifically, examining insulin along with body composition and other biomarkers of health would present greater implications to overall markers of health and performance.

Effects of a Low Carbohydrate Diet on Resting Testosterone

Testosterone is an anabolic hormone primarily released from the testes in men and in smaller amounts the ovaries in females. The primary purpose of testosterone is to synthesize stimulation of bone and muscle synthesis. Therefore, it is a popular hormone studied in the field of sports nutrition and specifically, its response to nutrition. As addressed earlier in this review, resting testosterone can be changed due to multiple different RT factors (Vingren et al., 2010). However, testosterone can be influenced by diet due to it being a steroid hormone synthesized from cholesterol (Vingren et al., 2010). Consuming a LCD specifically a LCD with an increase in dietary fat can lead to greater bioavailability of lipids for testosterone. Recently, Wilson et al. (2017) observed significant increases in testosterone levels following a 10 week RT program and ketogenic diet. However, resting testosterone levels have not been shown to increase following a ketogenic diet in healthy-weight men (Volek et al., 2002) and off-road

cyclists (Zajac et al., 2014). At this time it is not fully understood if healthy populations can increase resting testosterone levels while consuming a LCD high in fat, without RT interacting to increase resting testosterone levels.

Protein and Fat Consumption in Low Carbohydrate Diets

A decrease in CHO intake, subsequently leads to an increase in fats or proteins to maintain daily caloric intake. The specific macronutrient breakdown of ketogenic diets was discussed earlier, but a decrease in CHO intake can also lead to an increase in protein consumption. Protein intakes for athletes have been recommended to be 1.3-1.8 g/kg/day to maximize muscle protein synthesis and larger intakes (~2-3 g/kg/day) may be beneficial during periods of caloric restriction (Jager et al., 2017; Phillips & Van Loon, 2011). Individuals may want to increase dietary protein when on a low CHO diet to maintain caloric intake, satiety, and the high thermic effect of protein. A series of investigations by Antonio and colleagues suggests chronic protein intakes of greater than 2.5 g/kg/day do not negatively impact health or performance outcomes following RT (Antonio, Ellerbroek, Silver, Vargas, & Peacock, 2016; Antonio et al., 2016; Antonio, Peacock, Ellerbroek, Fromhoff, & Silver, 2014), but provide no performance or lean body mass adaptations compared to lower protein intakes. Variations in training goals and dietary goals may influence macronutrient distributions when LCD are employed, but evidence suggest it is safe to increase fat and protein intake when maintaining an isocaloric state.

Carbohydrate Supplementation in Resistance Training

Carbohydrate supplementation has become a popular ergogenic aid for multiple types of sporting events. Due to CHO being the predominant and preferred fuel during exercise, research examining the efficacy of CHO during exercise has also become very popular. Currently, it is suggested that during endurance exercise lasting from one to three hours and beyond, athletes ingest 30 to 90 $g \cdot hr^{-1}$ with the main goal of providing an exogenous fuel source to possible delay fatigue due to reductions in blood glucose, decreases in central nervous system drive, and reductions in muscle glycogen (Burke et al., 2011). However, less attention has been given to exercise such as RT and HIIT.

Effects on Resistance Training Performance

The efficacy of CHO supplementation surrounding RT has been extensively studied. However, there is a disparity in the literature regarding the beneficial effects of CHO. Performance benefits with CHO supplementation and resistance exercise have been documented in several investigations (Haff et al., 2001; Haff et al., 1999; Krings et al., 2016; Lambert et al., 1991; Oliver et al., 2016; Wax, Brown, Webb, & Kavazis, 2012; Wax, Kavazis, & Brown, 2013; Wax, Kinzey, Lyons, & Brown, 2010). Conversely, numerous investigations have failed to demonstrate performance benefits (Conley et al., 1995; Dalton et al., 1999; Fairchild et al., 2016; Haff et al., 2000; Kulik et al., 2008; Raposo, 2011; Rountree et al., 2017; Smith et al., 2017a; Vincent et al., 1993). Differences in performance outcomes may be attributed to type of resistance exercise, overall volume, and length of exercise protocol (Conley & Stone, 1996; Haff, Lehmkuhl, McCoy, & Stone, 2003).

During resistance exercise bouts, a primary source of fuel is muscle glycogen. Reductions in muscle glycogen have been observed in multiple RT protocols (Haff et al., 2000; Koopman et al., 2006; Macdougall et al., 1999; Pascoe et al., 1993; Robergs et al., 1991; Tesch et al., 1986; Tesch et al., 1998). Furthermore, Leveritt and Abernethy (1999) examined the effect of CHO depletion on subsequent strength performance and observed significant decrements in muscular strength (Leveritt & Abernethy, 1999). Increases in blood glucose levels with CHO supplementation (Haff et al., 1999; Lambert et al., 1991; Wax et al., 2013) may attenuate muscle glycogen stores to improve performance. However, recent evidence suggests no performance benefits even though blood glucose levels are elevated following CHO ingestion during RT (Fairchild et al., 2016). Blood glucose has been suggested to account for 10-25% of total metabolism during glycolysis (Brooks, Fahey, & Baldwin, 2005), therefore providing an exogenous CHO source may possibly reduce the chance of becoming hypoglycemic and/or increase CHO availability for substrate utilization in the muscle.

Training volume and training session length may be the determining factor in stimulating a demanding glycolytic effect for exogenous CHO to support RT performance. Recently, during a research study examining the effects of a full body strength and conditioning protocol, lasting 71 min, significant increases in performance was observed when ingesting CHO (Krings et al., 2016). These results are in agreement with other investigations lasting 56 min (Lambert et al., 1991), 57 min (Haff et al., 2001), 77 min (Haff et al., 1999). Shorter duration protocols have failed to demonstrate improvements in performance (Haff et al., 2000; Kulik et al., 2008). Similar to exogenous CHO supplementation recommendations during endurance exercise (Burke et al., 2011),

RT performance benefits may only be observed during training lasting one hour or longer.

A possible explanation of CHO supplementation improving performance during resistance exercise lasting under 60 minutes may be due to increases in central nervous system drive. It has been proposed swishing a CHO rich solution in the oral cavity can stimulate the reward centers in the brain (Chambers, Bridge, & Jones, 2009) and enhance activation of the areas involved in motor performance and sensory perception (Turner, Byblow, Stinear, & Gant, 2014). Jensen, Stellingwerff, and Klimstra (2015) observed beneficial results from CHO mouth rinse during lower body isometric knee extensions, when completed in a fatigued state. Recently, beneficial effects with CHO mouth rinse have been observed during bench press, back squat, (Clarke, Hammond, Kornilios, & Mundy, 2017) and isokinetic exercise (Bazzucchi, Patrizio, Felici, Nicolo, & Sacchetti, 2016). However, CHO mouth rinse has also been found to not improve maximal muscular strength and muscular endurance (Clarke, Kornilios, & Richardson, 2015; Dunkin & Phillips, 2017; Painelli et al., 2011). Exogenous CHO availability for muscular CHO metabolism and sensing of glucose receptors in the oral cavity may dependently or independently influence resistance exercise performance following CHO ingestion.

Carbohydrate Dosages During Resistance Training

Variations in dosages may partially explain the acute effects of CHO ingestion during RT. Investigations have supplemented participants with beverages containing 10% (Lambert et al., 1991; Oliver et al., 2016; Raposo, 2011; Wax et al., 2012; Wax et al., 2013; Wax et al., 2010), 20% (Haff et al., 2000; Haff et al., 2001; Haff et al., 1999; Kulik et al., 2008), 21% (Vincent et al., 1993), and 27% (Fairchild et al., 2016) CHO solutions.

However, it is generally accepted that CHO containing beverages formulated to supplement during exercise should contain <10% CHO concentrations and beverages containing >10% concentrations are utilized for CHO loading goals (Coombes & Hamilton, 2000). With limited research on gastric emptying and intestinal absorption during RT, extrapolations from endurance exercise need to be made. Lower concentrations of CHO content empty from the stomach at faster rates, but higher concentrations deliver similar amounts of CHO during early exercise and at greater rates later in exercise (Vist & Maughan, 1995). One negative impact of high CHO concentrated beverages is a decrease in water absorption (Noakes, Rehrer, & Maughan, 1991), potentially leading to negative effects associated with dehydration. Higher CHO beverages may deliver similar amounts of energy after an extended period of time, but during high intensity exercise research suggest significant impairments in gastric emptying when exercise intensity is above 70% $\dot{V}O_{2\,max}$ (Costill & Saltin, 1974; Leiper, Broad, & Maughan, 2001; Leiper, Nicholas, Ali, Williams, & Maughan, 2005) and during sports, such as soccer (Leiper, Prentice, Wrightson, & Maughan, 2001). Therefore, the high intensity nature of RT may partially explain the varying results with CHO supplementation.

To date, only one study has examined the effects of lower concentration CHO beverages during strength and conditioning sessions that included RT exercises. Krings et al. (2016) examined the effects of 3, 6, and 12% CHO solutions during a strength and conditioning session and concluded beverages of 3 and 6% CHO to be most beneficial for performance. Supplementing with 3 to 6% CHO compared to 12% CHO solutions may have allowed for CHO to be readily available for in the blood stream. Although this is an

assumption since blood glucose was not directly measured. This may be due to the reasons alluded to above: decrease in gastric emptying rates with higher intensity exercise and increase in emptying rates with lower CHO solutions. Sports beverage companies CHO beverages typically deliver 6-8% CHO in solution (Coombes & Hamilton, 2000) and research supports this range of concentrations during endurance exercise (Smith et al., 2013). Therefore, it may be more realistic for individuals completing RT with exogenous CHO supplementation to follow guidelines similar to endurance fueling recommendations (Burke et al., 2011). CHO beverages containing >10% solutions may be more beneficial for promoting recovery and muscle glycogen resynthesis following exercise.

Carbohydrate Supplementation in HIIT

Much like CHO ingestion during resistance and aerobic exercise, research has been directed towards the possible ergogenic effects of CHO during HIIT. CHO supplementation during HIIT may potentially minimize fatigue and improve performance. Several investigations have examined the efficacy of CHO ingestion during some form of HIIT (Khorshidi-Hosseini & Nakhostin-Roohi, 2013; Krings et al., 2017; Lee et al., 2014; Pomportes et al., 2016). CHO ingestion has been found to improve peak power output during cycling based repeated sprints (Lee et al., 2014; Pomportes et al., 2016), but not during a running anaerobic repeat sprint protocol (Khorshidi-Hosseini & Nakhostin-Roohi, 2013). Although there is minimal research examining HIIT protocol and CHO ingestion, several investigations have examined the effects of CHO mouth rinse on HIIT. Due to the high intensity nature of repeated sprints, CHO mouth rinse may be an

alternative to minimize gastrointestinal distress (de Oliveira & Burini, 2014) and still improve performance through non-metabolic pathways.

The CHO mouth rinse technique has been found to significantly improve peak power output in the first sprint of five cycling sprints (Beaven, Maulder, Pooley, Kilduff, & Cook, 2013), but not performance during repeated 20 m (Dorling & Earnest, 2013) and 40 m sprints (Bortolotti, Pereira, Oliveira, Cyrino, & Altimari, 2013). Due to the discrepancy in the literature, Krings et al. (2017) recently examined the effects of both CHO ingestion and mouth rise during five repeated 15 s maximal cycling sprints. The authors concluded CHO ingestion was the most beneficial treatment compared to ingesting a placebo and mouth rinsing either a CHO or placebo solution (Krings et al., 2017). Based on the current literature, CHO ingestion or mouth rinse is not likely to be detrimental to performance during exercise, such as HIIT. However, the effects of chronically ingesting CHO during HIIT are unknown.

Carbohydrate Dosages During HIIT

CHO containing beverages in investigations using some form of HIIT administered dosages of 20% (Khorshidi-Hosseini & Nakhostin-Roohi, 2013), 10% (Krings et al., 2017), 9% (Lee et al., 2014), and 7% (Pomportes et al., 2016). As stated earlier in this review, it is recommended that CHO containing beverages not exceed 10% solutions (Coombes & Hamilton, 2000). Interestingly, beverages administering CHO concentrations of 10% and below (Krings et al., 2017; Lee et al., 2014; Pomportes et al., 2016) observed significant performance benefits compared to a 20% concentration (Khorshidi-Hosseini & Nakhostin-Roohi, 2013).

Gastrointestinal Distress During Exercise

Gastrointestinal distress (GID) is a participant measure utilizing a likert scale, examining the impact of nutrition on symptoms of upper and lower GID during exercise. GID is typically reported in endurance athletes with complaints of GID being induced from physiological, mechanical, or nutritional means (de Oliveira, Burini, & Jeukendrup, 2014). Mechanical factors that can increase GID are related to body positioning and mechanical force produced during exercise. Running induces more GID than cycling and aggressive cycling positions (i.e. aero positioning) can induced greater GID compared to traditional seating positions (Peters et al., 2000). Furthermore, exercise intensity also plays a role GID. Increases in exercise intensity decreases blood flow to the gastrointestinal organs and increases blood flow to the periphery (i.e. skin, working muscles) (Qamar & Read, 1987). This would in turn increase the time it takes to empty the gut and delay the physiological effects for the nutritional supplement.

Although GID is typically only reported in endurance athletes, there is a limited amount of research with RT. Due to the mechanical forces imposed on the body when completing dynamic upper and lower body RT, GID may be an issue. However, without literature to support this claim, this is mere speculation. Content of a consumed beverage also effects feelings of GID. Compared to water, CHO rich beverages increase ratings of reflux, abdominal pain, intestinal cramps, and gas/flatulence during endurance exercise (van Nieuwenhoven, Brouns, & Kovacs, 2005). Measuring GID during exercise can be important to track both acute and chronic incidence rates. Overtime, reductions in GID could mean that an individual has improved their bodies ability to handles fluids and nutrients, but more specifically trained the gut (Jeukendrup, 2017). Jeukendrup (2017)

suggests that the gut is an important organ and can be trained like any other exercise organ. Monitoring changes in GID could be an indirect way to measure increases in gastric emptying. Specifically, when consuming CHO-rich beverages during exercise, reductions in GID could indicate greater CHO availability from the ingested beverage during exercise. Training the gut is a goal for many endurance athletes who rely on exogenous CHO supplementation during exercise but has received little interest with RT individuals. Considering the popularity of ergogenic aids consumed during RT (i.e. amino acids, pre-workouts, CHO), research in needed to establish nutrient and exercise induced GID during high-intensity exercise.

CHAPTER III

METHODS

Participants

Twenty-eight healthy males were recruited to participate in this investigation. Participants had to meet the following inclusion criteria: (1) meet the intermediate RT experience classification per the National Strength and Conditioning Association (NSCA; Haff & Triplett, 2016), (2) not currently taking any banned substances (steroids, etc.), (3) refrain from taking caffeinated supplements (thermogenics, pre-workouts), excluding traditional soda and coffee, for the duration of the study, (4) be classified as low risk assessed by a physical activity readiness questionnaire (PAR-Q), (5) meet minimum physical activity guidelines according to the ACSM (Medicine, 2013), (6) consume less than 40% of their current diet from fat. Participants were recruited from Mississippi State University via word of mouth, research flyers, and social media. Participants were required to attend 90% of the training sessions. Missing more than three training sessions led to exclusion from the study. Four participants were excluded from the study after the initial dietary screening due to habitually consuming more than 40% of their diet from fat. Three participants had to discontinue participation before beginning the 2-week familiarization period due to scheduling conflicts, one participant had to discontinue participation do to an injury sustained during strength max out testing, and two participants were unable to adhere to the diet before beginning the 4-week intervention

period. Therefore, a total of 18 subjects competed the study (supplemented group (n = 9), age = 21.0 ± 1.9 years, height = 179.2 ± 7.1, weight = 86.71 ± 9.62: non-supplemented group (n = 9), age = 19.7 ± 1.0 years, height = 179.3 ± 6.4, weight = 83.80 ± 12.62). Over 27 training sessions (familiarization plus experimental intervention), attendance for the supplemented (SUPP) group was 98.8% and the non-supplemented (NONSUPP) group was 98.0%. Attendance, including only the 20 training sessions during the experimental intervention, was 98.9% for the SUPP group and 97.2% for the NONSUPP group.

Experimental Design

The purpose of this study was to examine the effects of CHO timing during a RT and HIIT program while consuming a low CHO, high fat diet (LCHF). An overview of the study is shown in Figure 1. During the initial visit, participants were informed of the purpose and details of the study, gave written informed consent in accordance with Mississippi State University's Institutional Review Board, completed a general health history questionnaire, lifestyle evaluation questionnaire, PAR-Q, and were instructed to complete a 3-day dietary recall. Following this initial session, participants completed four additional pre-training sessions. Collection of height and mass and a Wingate anaerobic test (WaNT) were completed during Session 2. During Session 3, participants completed a familiarization with the peak oxygen consumption ($\dot{V}O_{2\ peak}$) test. Session 4 served as upper body muscular strength testing and session five served as lower body muscular strength testing. Sessions 4 and 5 were separated by at least two days. Upon completion of pre-training testing, participants completed a 6-week high-intensity training program. The first 2 weeks served as familiarization to RT and HIIT with no dietary intervention,

besides removing caffeinated supplements. Following 2 weeks of familiarization training, participants completed three more testing sessions: (1) a baseline blood draw and $\dot{V}O_{2\ peak}$ test and (2) body composition (BodPod and ultrasound) assessments. The final mid-testing session served as an informative meeting, notifying participants of their experimental groupings and providing nutritional information to help them adhere to the diet. During the last 4 weeks of training, participants completed RT 3 days per week and HIIT 2 days per week. Participants were randomly assigned to either train with CHO supplementation during and after training (SUPP; $n = 9$) group or train with no CHO supplementation during and after training (NONSUPP; $n = 9$). No other form of supplementation pre- and post-one hour of exercise was allowed and both groups had their daily dietary CHO intakes set at 2 $g \cdot kg^{-1} day^{-1}$ with the only difference being the SUPP group receiving 30 g of CHO during exercise and 40 g CHO immediately after exercise while the NONSUPP group spread the same CHO intake across the day. Every Friday of the 4-week training/nutrition intervention, blood glucose was measured three times via finger stick, for a total of 12 measurements. To examine the impact of ingesting flavored beverages during RT and HIIT sessions, participants reported GID pre and post the same RT and HIIT each week. Upon completion of the 6-week intervention, participants completed post-testing. Post-testing consisted of the same tests completed during both pre-testing and mid-testing: body composition (BodPod and ultrasound), WaNT, a baseline blood draw and $\dot{V}O_{2\ peak}$ test, maximal bench press strength, and maximal back squat strength. Participants were asked to maintain the same dietary habits from the last 4 weeks of the intervention during the post-testing week. In total participants completed 39 sessions. Due to the inability to blind the two experimental

groups, this study was not blinded to the participants nor the investigators. To minimize participant bias, the NONSUPP group received an artificially flavored beverage during exercise, with similar taste and scent to the beverages the SUPP group received.

Training Protocol

Following pre-testing, participants completed 2 weeks of training consisting of RT 3 days per week, for a total of 6 training sessions, and one familiarization session of HIIT. Upon completion of mid-testing, participants were randomly divided into two experimental groups, SUPP and NONSUPP. The groups were matched for age, mass, muscular strength (both bench press and back squat), dietary intake (daily kilocalorie intake), $\dot{V}O_{2\ peak}$, and mean power output. Participants then completed a 4-week supervised high-intensity training program. This consisted of RT 3 days per week and HIIT 2 days per week, for a total of 20 training sessions. During RT days, participants reported to the Strength and Conditioning Room (132B) in the Joseph A. Chromiak Applied Physiology Laboratory located in McCarthy Gymnasium. All HIIT sessions took place in the Cycling Room at the Joe Frank Sanderson Center. Participants trained in groups of 1-3 during RT sessions and in groups of 5-7 during HIIT sessions. Training sessions took place during the morning, afternoon, and night with participants being required to train at the same time of day for the duration of the study.

Resistance Training Protocol

The RT protocol followed a 6-week periodized training schedule. The protocol employed a linear periodization model, providing progressive overload across 6 weeks of training. The protocol is listed in Table 1. Weeks 1 and 2 served as lower intensity/higher

volume training to familiarize participants with the protocol. Following the first 2 weeks, repetitions decreased, and intensity increased for the final 4 weeks. A dynamic warmup was completed prior to beginning every RT session and was repeated each day. Participants were instructed to complete each exercise with proper form and were monitored by a NSCA Certified Strength and Conditioning Specialist during each session. Performance was measured during the final set of back squats, bench press, pushups, and pull-ups during the full-body training day each week (Friday). During each final set, participants were instructed to complete repetitions until volitional fatigue or deemed failure by a break in form assessed by the investigator. Volume from back squats, bench press, and total repetitions completed during pushups and pull-ups were used for data analysis. Prior to beginning the RT protocol, true one-repetition max (1-RM) for bench press and back squat was tested. Furthermore, 1-RM was predicted for the exercises of Romanian deadlift, Bulgarian split squats, lunges, bent-over row, upright row, shoulder press, and biceps curls.

High Intensity Interval Training

An overview of the HIIT sessions and training progressions are displayed in Table 2. The HIIT model utilized in the proposed study was adopted from previous investigations utilizing repeated bouts of maximal sprints lasting 30 s interspersed with active recovery periods (Burgomaster et al., 2006; Burgomaster et al., 2005). Prior to beginning each HIIT session, participants were fitted with heart rate (HR) monitors (H10, Polar Electro Inc., Lake Success, NY, USA). Participants were then assigned to a spin bike (Group Cycle Ride, TechnoGym, USA Corp., West, NJ, USA). Saddle height of the bike was adjusted for each participant to ensure 5-10° of knee flexion while the foot was

in the lowest position (Wilson et al., 2017). Seat position was noted and kept the same for all training sessions. Due to the inability of spin bikes to measure work output, HR was measured to monitor intensity throughout each session. Heart rate was monitored using a HR response software (Team Polar, Polar Electro Inc., Lake Success, NY, USA) feeding live data to an iPad (Apple, Cupertino, CA, USA). During each sprint, participants were instructed to spin as hard as possible against a self-selected resistance and achieve an intensity of 90% maximum HR (HR_{max}). The intensity was established off the highest HR_{max} achieved from the first two $\dot{V}O_{2\ peak}$ tests. If 90% HR_{max} was not reached during a sprint, the participant was instructed to increase the resistance or pedal cadence of the spin bike. Since there were multiple participants in each spin class, immediately following each sprint a screen shot was taken on the iPad to measure intensity.

Experimental Procedures

Peak Oxygen Consumption Test

Each $\dot{V}O_{2\ peak}$ test was conducted on a cycle ergometer (Velotron, Seattle, WA, USA). During the familiarization $\dot{V}O_{2\ peak}$ test, participants were not instructed to fast, however, during the pre-and post-experimental test participants were instructed to arrive with at least a 10-hour fast. Participants begin the test at 100 W for three minutes. Following the first 3-min stage, there were three 3-min stages. These stages corresponded to 150, 200, and 250 W. Following the first four stages, each subsequent stage increased by 25 W per minute. The test was concluded when participants stopped due to volitional fatigue or the participant could not maintain a cadence of 50 RPM. The cardiorespiratory responses of HR, $\dot{V}O_2$, volume of carbon dioxide expenditure ($\dot{V}CO_2$), ventilation ($\dot{V}E$), breathing rate (BR) and respiratory exchange rate (RER) were collected for the duration

of the test. Furthermore, subjective fatigue was measured 30 s prior to beginning a new stage using the 0-10 modified Borg rating of perceived exertion scale (Borg, 1982).

HR response was measured 30 s prior to beginning a new stage using a HR monitor (FT1, Polar Electro Inc., Lake Success, NY, USA). Furthermore, blood lactate was measured at the same time increments as RPE and HR via fingerstick technique. A small blood sample (0.3 μL) was collected during each fingerstick and measured using a portable lactate analyzer (Lactate Pro 2, Arkray, Japan). $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and RER were measured using a MOXUS metabolic cart (AEI Technologies, Pittsburgh, PA). Air volume and flow rate were measured using a pulmonary pneumotachometer (AEI Technologies, Pittsburgh, PA). In addition, a S-3A/I and CD-3A analyzer (AEI Technologies, Pittsburgh, PA) were used to separately measure $\dot{V}O_2$ and $\dot{V}CO_2$.

Body Composition

Body mass and height were measured using a digital scale (Defender 5000, Ohaus Corporation, Parsippany, NJ, USA) and digital stadiometer (235D; QuickMedical, Issaquah, WA, USA). To track body mass changes throughout the 4-week intervention period, body mass was measured prior to each RT session, for a total of 12 measurements. Pre-and post-fat mass, fat-free mass, and % body fat were determined via air displacement plethysmography (BodPod, COSMED USA, Concord, CA, USA). All testing took place in accordance with the manufacturer's instructions. This included participants withholding from drinking alcohol 48 h prior to testing and fasting at least four hours before testing. Participants also completed testing at the same time of day during pre- and post-testing and wore tight fitting spandex undergarments and a swim cap. Thoracic gas was estimated by the BodPod.

An assessment of muscle cross sectional area (CSA) was conducted to determine muscle thickness via ultrasound (LOGIQ e Diagnostic Ultrasound System, General Electric, Wauwatosa, WI, USA). A transducer with an imaging frequency bandwidth of 5.0-23.0 MHz and 12.7 x 53 mm footprint was used to measure CSA of the biceps, triceps, gastrocnemius, hamstrings, and quadriceps. Each muscle was measured three times and the average was used for data analysis. To ensure proper CSA measurements across all participants, the following standardized sites were used. The triceps was measured at 60% of the distance from the olecranon process to the acromion process along the muscle belly. Directly horizontal from the triceps site, the biceps site was measured. During measurement of the triceps and biceps, participants were standing, relaxed, and rested their arm to the side with their elbow extended at a comfortable position. Upon marking both anatomical sites, the distance from the edge of the muscle belly to the humerus comprises the CSA of each muscle (Smith et al., 2017a). During measurement of the quadriceps, participants laid on their non-dominant side, with their knee slightly flexed. The site used was 50% of the distance from lateral condyle of the tibia and the greater trochanter of the femur on the lateral side of the quadriceps (Suchomel & Stone, 2017). For measurement of the gastrocnemius, participants lied prone with their ankle relaxed and dorsiflexed. The middle of the distance between the proximal and distal tendon insertion on the midsagittal plane of the gastrocnemius (Morse et al., 2008). The hamstrings were measured at the 50% distance of the posterior aspect of the fibular head and ischial tuberosity while participants lied prone relaxing on a training table (Suchomel & Stone, 2017). All measurements were marked with a permanent marker on the dominant side of the body and made by the same investigator. To ensure

participants kept their sites marked throughout the 4-week intervention, permanent markers were provided during each RT session. Investigators also constantly reminded participants to continue re-marking their sites.

Muscular Strength Testing

True and estimated 1-RM testing procedures were completed according to guidelines put forth by the NSCA (Haff & Triplett, 2015). True 1-RM was obtained for bench press and back squat. During true 1-RM testing, participant completed a warmup set with a light resistance for 5-10 repetitions followed by 1-min rest. Depending on the exercise, 4.5-18 kg (10-40 lb) was added to the barbell and 3-5 repetitions were completed, followed by two minutes of rest. 4.5-18 kg (10-40 lb) were again added to the bar and 3-5 repetitions were completed, followed by 2 min of rest. After the final warmup set, participants were instructed to lift a 1-RM within 3-5 sets, with two minutes of rest between sets. For the exercises of Romanian deadlift, Bulgarian split squats, lunges, bent-over row, hammer curls, upright row, and shoulder press, a predicted 1-RM test with a target range of 2-5 maximum repetitions was used. Repetitions and weight lifted were used to predict 1-RM (Brzycki, 1993). During predicted 1-RM testing, a three-set approach was used [one warm-up with light weight (5-10 repetitions), one warmup with 50-80% perceived 1-RM (3-5 repetitions), and one max estimation set].

During upper body strength testing, exercises were completed in the following order: bench press, shoulder press, bent-over row, upright row, and hammer curls. Lower body testing took place in this order: back squat, Romanian deadlift, step-ups, Bulgarian Split squat, and lunges. After the 6-week intervention, participants only completed post-1-RM testing with the exercises of bench press and back squat.

Wingate Testing

WaNT took place on a cycle ergometer using Velotron™ Wingate software (RacerMate, Inc., Seattle, WA, USA). Prior to testing, saddle height of the ergometer was adjusted for each participant to ensure 5-10° knee flexion while foot is in the lowest position (Wilson et al., 2017). Seat position was noted and kept for pre-and post-testing. Participants then commenced a 5-min warm-up against a self-selected pace. Prior to beginning the test, there was a 30 s warm-up period. The first 20 s were completed against a resistance of 100 W and 10 s prior to beginning the test all the resistance was removed from the cycle ergometer. Six seconds prior to the load initiation, participants were instructed to maximally increase their cadence. A resistance of 0.075 $kg \cdot kg^{-1}$ body mass was magnetically added and participants were instructed to cycle with maximal effort for 30 s while keeping their buttock on the seat. Verbal encouragement was provided by the investigators during each test. Following completion of the test, a 5-min cool down was completed on either a cycle ergometer or treadmill. Absolute peak power output, absolute mean power output, relative mean power output (mean power/ body mass), relative peak power output (peak power/ body mass), total work, and fatigue index ((peak watts- minimum watts)/30 seconds) was measured using the Velotron™ Wingate software (Racermate, Inc., Seattle, WA, USA).

Gastrointestinal Distress Scale

GID was measured using a 10-point likert scale assessing feelings of nausea, regurgitation/reflux, stomach fullness, abdominal cramps, gas/flatulence, and urge to defectate. Participants were rated a scale of 0-10 with 0 representing no discomfort, 5 representing moderate discomfort, and 10 representing unbearable discomfort. This GID

scale has been previously used to assess exercise-associated GID during endurance exercise (Wilson, 2016, 2017). To familiarize participants with the GID scale, the investigators explained the ratings scale and symptoms during the initial meeting. GID was measured immediately before and after exercise. GID was measured each Monday lower-body RT session and Tuesday HIIT during the 4-week experimental intervention totaling eight sessions. By measuring GID pre- and post-exercise the investigators ensured the post-workout beverage would not influence ratings of GID obtained from drinking ~500 mL CHO and non-CHO during exercise for the SUPP and NONSUPP groups.

Blood Analysis

Intravenous blood samples were obtained after familiarization training and after 4 weeks of the training/nutritional intervention. Prior to commencing $\dot{V}O_{2\ peak}$ testing, subjects arrived at the exercise physiology lab following at least a 10-hour fast. While lying supine, 14 mL of blood where drawn from the antecubital vein via butterfly needle into an EDTA anticoagulant sealed vacutainer (7 mL) and a sodium heparin sealed vacutainer (7 mL). Blood samples were immediately centrifuged at 2500 rpm. Plasma was then aliquoted and stored in a -80ºC freezer until further analysis. Insulin (Eagle Bio, Nashua, NH, USA) was analyzed using a ELISA assay kit. The insulin assay used a sandwich technique and was analyzed using a iMark Bio-Rad microplate absorbance reader (Life Science Research, Hercules, California, USA), reading at an absorbance of 450 nm and reference of 655 nm using the endpoint technique. Results for insulin were calculated from the average absorbance of the duplicate results using a point-to-point standard curve fitting. Testosterone (Eagle Bio, Nashua, NH, USA) was analyzed using a

ELISA assay kit and competitive technique. The assay kit measured total testosterone concentration (free plus bound testosterone bound to sex-hormone binding globulin). Testosterone was analyzed using a microplate absorbance reader, reading at an absorbance of 450 nm and reference of 630 nm and using the endpoint method. Testosterone was calculated as the average absorbance of duplicate results using a four-parameter estimate. Glucose (Pointe Scientific, Canton, MI, USA) was analyzed using a liquid glucose oxidase reagent set. Glucose absorbance was measured using an endpoint method at a wavelength of 500 nm. A Pointe 180II spectrophotometer (Pointe Scientific, Canton, Michigan, USA) was used to read the absorbance and calculate glucose concentrations ((subject absorbance/standard absorbance) × concentration of glucose standard). Glucose samples were run in triplicates. Intra-assay coefficients of variation were 7.7%, 3.2% and 3.3% for insulin, testosterone, and glucose, respectively.

Blood glucose was also measured via finger stick on 12 occasions. During the 4-week training/nutrition intervention, blood samples were collected each Friday immediately before beginning exercise (pre-exercise), after the fourth exercise (mid exercise), and at the completion of exercise (post-exercise). The initial blood glucose droplet obtained following the fingerstick (~0.6 mμL) was wiped and discarded. A 0.6 μL sample was the collected and analyzed using a portable blood glucose monitoring system (Precision Xtra, Abbott Laboratories, Chicago, IL, USA).

Nutritional Intervention

Following the initial session and completion of the informed consent, participants were instructed to eat as they normally would and complete a three-day food log. All

food intake was logged into myfitnesspal (Under Armour, Baltimore, MD, USA). Upon completion of the three-day food log, participants sent dietary information to the primary investigator and the food was analyzed using Nutrionist-Pro (version 2.2, 2005, Axxya Systems-Nutrionist Pro, Stafford, TX. USA). The purpose of a baseline nutrition evaluation was to establish current daily macronutrient breakdown and total kilocalorie (kcal) intake. After the initial dietary collection, participants were instructed to maintain their habitual diets until the beginning of the 4-week exercise/dietary intervention. Upon beginning the 4-week intervention, the investigators provided each participant with a specific kcal intake and macronutrient breakdown: 2 $g \cdot kg^{-1} \cdot day^{-1}$ CHO, 2 $g \cdot kg^{-1} \cdot day^{-1}$ protein (PRO), and the remaining kcals from fat. The specific kcal intake was recommended to ensure that participants received ~25% caloric intake from CHO and PRO, and ~50% from fat. Participants were instructed to try and intake as close to their kcal guidelines but given a ± 250 kcals range. To ensure dietary guidelines were being met during the dietary intervention, participants were asked to log their food intake every day and provide their food logs to the principal investigator on every Tuesday and Friday, which were dietary intakes from Mondays and Thursdays. On days that participants were not sending their food logs to the principal investigator, random diet checks occurred. Each participant, over the course of the 4-week experimental intervention was randomly checked on 2 weekdays and 2 weekend days.

During the final mid-testing session, participants were informed of their experimental grouping and given specific dietary guidelines. They were also given examples of sample meals plans and restaurant meal plans to help adherence with the diet. Also, at this time each participant received 10 prepackaged whey isolate PRO

packets (ISO-100, Dymatize Enterprises, LLC, Dallas, TX, USA). Following the first 2 weeks of the experimental intervention, 10 more packets were given. Each packet contained 25 g of PRO.

On the first Monday of the 4-week experimental intervention, the specific dietary guidelines listed above began. However, there were two different weekday CHO timing schedules for each group. Both groups were instructed to ingest a PRO shake containing 4-8 oz. of water and their provided prepackaged PRO powder one hour before commencing each training session. Following ingestion of the PRO shake, participants were not allowed to consume any other nutritious items besides water prior to beginning training sessions. Immediately upon beginning both the RT and HIIT sessions, the SUPP group was given a shaker bottle containing ~500 mL of water and 30 g of a 2:1 (glucose: fructose) CHO equating to a 6% CHO beverage. This concentration of beverage is used in common sports drinks (Coombes & Hamilton, 2000) and was recently found to be beneficial during bouts of high intensity strength and conditioning (Krings et al., 2016). The NONSUPP group was given a beverage consisting of the same fluid volume but with an artificial flavor matching the SUPP groups beverage containing no nutritional value. Both beverages were lemon-lime flavored. During each training session, a specific drinking schedule was followed for both groups to ensure that each beverage was consumed similarly throughout each session. During RT sessions, 100 mL of the experimental beverage was ingested after the warm-up, and after the final set of each of the first four exercises. During the first 2 weeks of HIIT, 100 mL of the experimental beverage was ingested before beginning the warmup, and one minute prior to beginning the each of the four sprints. During the final 2 HIIT weeks, 100 mL of the experimental

beverage was ingested one minute prior to beginning sprints 1-5. During the familiarization training, the same intra-workout schedule was utilized, but with water, to familiarize participants with fluid volumes. Following completion of RT and HIIT sessions, both groups received a beverage containing 25 g of PRO (Elite Whey, Dymatize Enterprises, LLC, Dallas, TX, USA). However, the SUPP groups beverage also contained 40 g of maltodextrin (Carbo Gain, NOW FOODS, Bloomington, IL, USA). Total fluid volume for both post-exercise beverages were 400 mL delivering a 10% CHO solution for the SUPP group. All intra-beverages were created and packaged by Dymatize Enterprises, LLC (Dallas, TX) for the purposes of this study and delivered to the principal investigator prior to beginning the study. Furthermore, both groups were instructed to enter their specific intra and post-exercise nutrition into their dietary logs at the beginning of each day.

Since the purpose of this study was to examine the effects of CHO timing during a high-intensity training program, specific daily nutrition guidelines were set. Most importantly, participants in both groups were not be allowed to ingest CHO for the first hour after exercise, besides for the SUPP groups post-workout CHO containing beverage. This guideline was put into place to ensure that the NONSUPP group was not taking advantage of the initial rapid glycogen resynthesis period after strenuous glycogen depleting exercise (Jentjens & Jeukendrup, 2003). Outside of the one-hour period pre- and post-exercise, participants were instructed to distribute macronutrient intake and kcals equally throughout the day. The authors accept that a limitation of this investigation is the timing and intake of nutrition outside of each training session. However, the primary aim of this investigation was to exam the influence of CHO ingestion during and

immediately after exercise, so maintaining guided CHO intake for the remainder of each day will answer the proposed research question.

Upon completion of the 4-week training/nutrition intervention each participant was instructed to maintain the same dietary recommendations laid forth during the study. Participants completed the diet and the investigation following the final post-testing session (lower body maximal strength testing). Overall participants completed the dietary intervention for 34-35 days.

Data Analysis

During the initial habitual diet collection, participants reported diets for three days. Those three days were averaged together and used for data analysis. During the 4-week dietary/training intervention, diets were collected twice per week and the average of both days was used or data analysis. The dietary variables used for data analysis included absolute gram intake per day, relative intake per day ($g \cdot kg^{-1}$), percentage of calories coining from the specific macronutrient. Total kcal intake per day was also used for data analysis. Body mass was measured 3 days/week during the experimental intervention and averaged to represent body mass across each of the 4 weeks.

Data were recorded during the $\dot{V}O_{2\ peak}$ test with breath by breath averages. Due to the variation in time to exhaustion (TTE) on the $\dot{V}O_{2\ peak}$ test, data were analyzed from only the first three stages of the test for BR, $\dot{V}E$, and RER. BR, $\dot{V}E$, and RER were analyzed by averaging breath by breath results across the final 30 seconds of each of the first three stages. Blood lactate measured during the first three stages of the $\dot{V}O_{2\ peak}$ test and at immediately post-test was used for data analysis. HR and RPE measured during

the first three stages and post-test was used for analysis. Absolute and relative $\dot{V}O_{2\ peak}$ were reported as the highest 30 second average achieved during the $\dot{V}O_{2\ peak}$ test.

Volume of total work for back squat, bench press, pushups, and pull-ups were collected each Friday of the 4-week intervention. Back squat and bench press volume were calculated as final set mass × repetitions. Pushup and pull-up volume was calculated as sets × repetitions.

Statistical Methods

All data are reported as mean ± standard deviation and with an alpha level set a priori at $p \leq 0.05$. Baseline characteristic comparisons between groups were analyzed using an independent samples *t*-test. Baseline characteristics are presented in Table 3. The dependent variables from the WaNT, absolute and relative $\dot{V}O_{2\ peak}$, TTE during the $\dot{V}O_{2\ peak}$ test, testosterone, blood glucose (resting values), insulin, maximal strength (bench press and back squat 1-RM), and body composition (% body fat, fat free mass, and fat mass, ultrasound) were analyzed using a two-way repeated measure analysis of variance (RMANOVA; group × time). Body mass and RT volume (bench press, back squat, pushups, pull-ups) were analyzed using a two-way RMANOVA (group × week). Results from the $\dot{V}O_{2\ peak}$ test, including: BR, $\dot{V}E$, HR, RER, blood lactate, and RPE were analyzed using a three-way RMANOVA (group x time x stage). Blood glucose measured before, during, and after each Friday RT session and was analyzed using a three-way RMANOVA (group × time × week). Gastrointestinal distress data were measured using a four-way RMANOVA (exercise × group × time × week). Bonferroni's post hoc test was used in instance of a significant main effect or interaction effect. Partial eta squared was

calculated for all RMANOVAs. Partial eta squared represents the proportion of total variance explained by treatment effects and is used to interpret meaningful differences. Interpretations are presented as: η_p^2: 0.2 = small effect; η_p^2: 0.5 = moderate effect; η_p^2: 0.8 = large effect (Cohen, 1988). All statistical analysis was completed using SPSS (Version 24, IBM Corporation, Armonk, NY, USA).

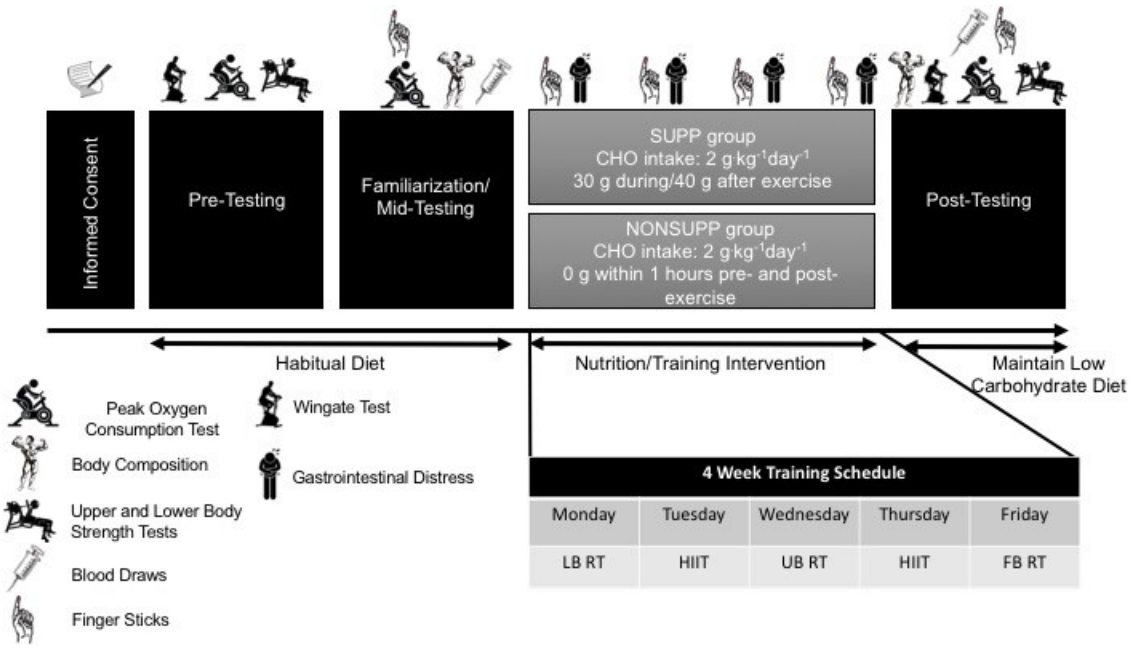

Figure 1. Schematic of Experimental Protocol. The figure represents the overall timeline of the investigation. SUPP= train with exogenous carbohydrate supplementation before and after group, NONSUPP= train without exogenous carbohydrate supplementation before and after group, UB= upper body, LB= lower body, RT= resistance training, HIIT= high-intensity interval training.

Table 1

Resistance Training Program

Monday **Lower Body**			
Exercise	Sets	Repetitions	Intensity
Back Squat†	3	12/8/6	67/80/85%
Romanian Deadlift†	3	12/8/6	67/80/85%
Step-Ups†	3	12/10/8	67/75/80%
Bulgarian Split Squats†	3	12/10/8	67/75/80%
Calf Raises^	3	12/15/20	Body Weight
Curl-ups*	2	20/25/30	Body Weight
Russian Twists*	2	20/25/30	Medicine Ball
Wednesday **Upper Body**			
Exercise	Sets	Reps	Intensity
Barbell Bench Press†	3	12/8/6	67/80/85%
Bent Over Row†	3	12/8/6	67/80/85%
Shoulder Press†	3	12/8/6	67/80/85%
Upright Row†	3	12/8/6	67/80/85%
(1) Hammer Curls^	3	12/8/6	67/80/85%
(2) Triceps Dips^	3	10/15/20	Body Weight
Curl-ups*	2	20/25/30	Body Weight
Planks*	2	45/60/75 secs	Body Weight
Friday **Full Body**			
Exercise	Sets	Reps	Intensity
Back Squat†	2/ final to failure	12/8/6	67/80/85%
Bench Press†	2/ final to failure	12/8/6	67/80/85%
Lunges†	3	12/10/8	67/75/80%
Shoulder Press†	3	12/8/6	67/80/85%
(1) Pushups^	3/ final to failure	10/15/20	Body Weight
(2) Pull-ups^	3/ final to failure	6/8/10	Body Weight
Planks*	2	45/60/75 secs	Body Weight
Side Planks*	2	30/45/60 secs	Body Weight

Exercises listed as (1) and (2) were completed as a superset; *30 seconds of rest between sets, ^60 seconds of rest between sets, †120 seconds of rest between sets. Hyphens between repetitions and intensity represent first two weeks/weeks three and four/ weeks five and six. Half of the repetitions were completed on each leg for the unilateral exercises (step-ups and Bulgarian split squats).

Table 2

High Intensity Interval Training Program

	Week 1	**Week 2**	**Week 3**	**Week 4**
Days per week	2 days	2 days	2 days	2 days
Volume	30 s x 4 sprints	30 s x 4 sprints	30 s x 5 sprints	30 s x 6 sprints
Rest Periods	4.5 minutes	4.5 minutes	4.5 minutes	4.5 minutes
Intensity	"All out" effort	"All out" effort	"All out" effort	"All out" effort
Heart Rate	~90 % HR max	~90 % HR max	~90 % HR max	~90 % HR max
Warm-up	5 minutes	5 minutes	5 minutes	5 minutes
Cool-down	5 minutes	5 minutes	5 minutes	5 minutes
Time per session	25.5 minutes	25.5 minutes	30.5 minutes	35 minutes
Time per week	51 minutes	51 minutes	61 minutes	70 minutes

This table represents the high intensity interval training program. The program is broken down into daily training time and weekly training time required across the four weeks of training.

CHAPTER IV

RESULTS

Anthropometrics

Body composition results are presented in Table 5. There was no group × week interaction effect for body mass (F = 0.15, $p = 0.78$, $\eta_p^2 = 0.009$) or differences in body between groups (F = 0.14, $p = 0.72$, $\eta_p^2 = 0.009$) and weeks (F = 0.16, $p = 0.78$, $\eta_p^2 = 0.010$). No group × time interaction effects were observed for percent body fat (F = 0.36, $p = 0.56$, $\eta_p^2 = 0.022$), fat mass (F = 0.84, $p = 0.37$, $\eta_p^2 = 0.050$), and fat free mass (F = 0.38, $p = 0.55$, $\eta_p^2 = 0.023$).There were also no differences between groups for percent body fat (F = 3.92, $p = 0.07$, $\eta_p^2 = 0.197$), fat mass (F = 3.65, $p = 0.07$, $\eta_p^2 = 0.186$), or fat free mass (F = 3.91, $p = 0.07$, $\eta_p^2 = 0.197$). There were also no changes in either the SUPP or NONSUPP groups percent body fat (F = 2.37, $p = 0.14$, $\eta_p^2 = 0.129$), fat mass (F = 1.59, $p = 0.23$, $\eta_p^2 = 0.090$), or fat free mass (F = 2.42, $p = 0.14$, $\eta_p^2 = 0.131$) as a result of the experimental intervention.

Ultrasonography

Ultrasound results are presented in Table 5. The data are presented as overall mean ± standard deviation for biceps, triceps, quadriceps, hamstrings, and gastrocnemius thicknesses. The coefficient of variation is reported for each muscle and time-point (pre and post) to examine the variability across three measurements.

Biceps

There was no group × time interaction effect (F = 0.35, $p = 0.56$, $\eta_p^2 = 0.021$) for biceps thickness. Biceps thickness was not different between groups (F = 0.21, $p = 0.66$, $\eta_p^2 = 0.013$). However, there was a significant increase in biceps thickness from pre-to post-intervention (F = 6.56, $p = 0.02$, $\eta_p^2 = 0.291$).

Triceps

No group × time interaction effect (F = 0.68, $p = 0.42$, $\eta_p^2 = 0.041$) was observed for triceps thickness. There were no differences between groups (F = 0.34, $p = 0.57$, $\eta_p^2 = 0.021$) or times (F = 0.97, $p = 0.34$, $\eta_p^2 = 0.057$) for triceps thickness.

Gastrocnemius

There was no group × time interaction effect (F = 0.29, $p = 0.57$, $\eta_p^2 = 0.021$) or differences between groups (F = 0.53, $p = 0.48$, $\eta_p^2 = 0.032$). Gastrocnemius thickness did not change as a result of the experimental intervention (F = 0.34, $p = 0.57$, $\eta_p^2 = 0.021$).

Quadriceps

There was also no group × time interaction effect (F = 0.02, $p = 0.90$, $\eta_p^2 = 0.001$) for quadriceps thickness. There were no significant differences in quadriceps thickness as a result of group (F = 0.07, $p = 0.78$, $\eta_p^2 = 0.004$) or time (F = 3.94, $p = 0.06$, $\eta_p^2 = 0.198$).

Hamstrings

No group × time interaction effect (F = 0.14, $p = 0.72$, $\eta_p^2 = 0.009$) was noted for hamstring thickness. Hamstring thickness did not change as a result of the experimental

intervention (F = 0.55, $p = 0.47$, $\eta_p^2 = 0.033$) and there was no difference between groups (F = 0.83, $p = 0.38$, $\eta_p^2 = 0.049$).

Macronutrient Diet Breakdown

Kilocalories

There was no group × week interaction effect (F = 0.73, $p = 0.49$, $\eta_p^2 = 0.043$) for kcals. Overall, there was no significant difference in kcal intake between the SUPP (2735.76 ± 408.73 kcals) and NONSUPP (2844.58 ± 353.65 kcals) groups across the duration of the study (F = 1.44, $p = 0.25$, $\eta_p^2 = 0.083$). However, kcal intake significantly differed across weeks (Habitual diet: 2472.18 ± 500.78 kcals, Week 1: 2880.31 ± 320.18 kcals, Week 2: 2865.00 ± 334.94 kcals, Week 3: 2821.56 ± 239.34 kcals, Week 4: 3008.44 ± 437.81, F = 7.28, $p = 0.002$, $\eta_p^2 = 0.329$). Habitual diet kcal intake was significantly lower than weeks one and four ($p < 0.05$), but not different from weeks two and three ($p > 0.05$). The goal of this study was to recommend participants to intake a greater number of kcals from their habitual diet, due to the increased exercise demands of the study. In total, 11 of the 18 participants were given a greater kcal intake from their habitual diet. This determination was made by taking the participants habitual diets and calculating 2 $g \cdot kg^{-1}$ CHO and PRO, and the remaining kcals coming from fat. Therefore, on average participants received ~25%, ~25%, and ~50% of their kcals from CHO, PRO, and fat.

Dietary Carbohydrate Intake

There was no group × week interaction effects for grams of CHO intake per day (F = 1.65, $p = 0.22$, $\eta_p^2 = 0.093$), relative CHO intake (F = 1.46, $p = 0.25$, $\eta_p^2 = 0.084$), or

percentage CHO intake (F = 0.83, $p = 0.44$, $\eta_p^2 = 0.049$). There were no differences between groups in regard to grams of CHO intake per day (F = 0.50, $p = 0.49$, $\eta_p^2 = 0.030$), relative CHO intake, expressed as $g \cdot kg^{-1}$ (F = 1.38, $p = 0.26$, $\eta_p^2 = 0.077$), and percentage of total daily dietary intake (F = 0.64, $p = 0.43$, $\eta_p^2 = 0.039$). there were no differences between groups. However, there was a significant main effect for CHO intake, expressed as total grams (F = 26.32, $p < 0.0001$, $\eta_p^2 = 0.622$), $g \cdot kg^{-1}$ (F = 22.2, $p < 0.0001$, $\eta_p^2 = 0.581$), and percentage intake (F = 105.21, $p < 0.0001$, $\eta_p^2 = 0.868$) between weeks. CHO intake was significantly greater during participants' habitual diet than weeks 1-4 of the experimental intervention ($p < 0.0001$). There were no differences in CHO intake between weeks 1-4 ($p > 0.05$). Dietary CHO intake is presented in Table 6.

Dietary Protein Intake

There was no group × week interaction effects for percentage of daily intake (F = 0.57, $p = 0.53$, $\eta_p^2 = 0.034$), total grams (F = 1.47, $p = 0.24$, $\eta_p^2 = 0.084$), and relative PRO intake (F = 2.41, $p = 0.09$, $\eta_p^2 = 0.131$). Daily PRO intake (percentage of daily intake (F = 0.05, $p = 0.83$, $\eta_p^2 = 0.003$), total grams (F = 0.99, $p = 0.33$, $\eta_p^2 = 0.058$), and relative PRO intake per day ($g \cdot kg^{-1}$) (F = 0.64, $p = 0.44$, $\eta_p^2 = 0.039$) were not different between groups. The percentage of PRO intake per day did not differ between weeks (F = 2.82, $p = 0.09$, $\eta_p^2 = 0.150$). There was a significant difference in grams of PRO per day (F = 22.18, $p < 0.0001$, $\eta_p^2 = 0.281$) and relative PRO per day (F = 15.87, $p = < 0.0001$, $\eta_p^2 = 0.495$) for time, with participants taking in a significantly lower number of grams of PRO during their habitual diet compared to weeks 1-4 ($p < 0.0001$). There were no differences in grams of PRO or relative PRO intake between weeks 1-4 ($p > 0.05$). Dietary PRO intake is presented in Table 7.

Dietary Fat Intake

Regarding fat intake, there were no group × week interaction effects for relative ($F = 0.62$, $p = 0.58$, $\eta_p^2 = 0.037$), absolute ($F = 05$, $p = 0.61$, $\eta_p^2 = 0.035$), and percentage of fat intake ($F = 0.21$, $p = 0.85$, $\eta_p^2 = 0.013$). There were no significant differences for relative ($g \cdot kg^{-1}$) fat intake ($F = 1.24$, $p = 0.28$, $\eta_p^2 = 0.072$), absolute ($g \cdot day^{-1}$) ($F = 1.42$, $p = 0.25$, $\eta_p^2 = 0.082$), or percentage of fat intake ($F = 1.18$, $p = 0.29$, $\eta_p^2 = 0.069$) for groups. However, there were significant differences in weeks for relative ($F = 50.94$, $p < 0.0001$, $\eta_p^2 = 0.761$), absolute ($F = 53.42$, $p < 0.0001$, $\eta_p^2 = 0.770$), and percentage of fat intake ($F = 68.03$, $p < 0.0001$, $\eta_p^2 = 0.810$) for groups. Fat intake significantly increased from the habitual diet to weeks 1-4 ($p < 0.0001$) with no differences between weeks 1-4 ($p > 0.05$). Dietary fat intake is presented in Table 8.

Wingate Testing

Mean Power Output

No group × time interaction effect was observed for absolute mean power output ($F = 1.31$, $p = 0.72$, $\eta_p^2 = 0.008$). However, absolute mean power output significantly increased as a result of the experimental intervention ($F = 14.61$, $p = 0.002$, $\eta_p^2 = 0.477$), but there were no differences between groups ($F = 0.11$, $p = 0.75$, $\eta_p^2 = 0.007$).

Regarding relative mean power output, no group × time interaction effect for relative mean power output ($F = 0.84$, $p = 0.37$, $\eta_p^2 = 0.050$) was noted. Relative mean power output increased as a result of the experimental intervention ($F = 8.46$, $p = 0.01$, $\eta_p^2 = 0.346$). There were no differences between groups ($F = 2.17$, $p = 0.16$, $\eta_p^2 = 0.120$).

Peak Power Output

There was no group × time interaction effect for absolute peak power output (F = 0.91, $p = 0.35$, $\eta_p^2 = 0.054$). There were also no differences in absolute peak power output between groups (F = 0.10, $p = 0.76$, $\eta_p^2 = 0.006$) or time (F = 0.73, $p = 0.41$, $\eta_p^2 = 0.044$).

No group × time interaction effect was noted for relative peak power output (F = 0.36, $p = 0.56$, $\eta_p^2 = 0.022$). Relative peak power was significantly greater after the experimental intervention (F = 34.88, $p < 0.0001$, $\eta_p^2 = 0.686$), with no differences between groups (F = 0.19, $p = 0.89$, $\eta_p^2 = 0.001$).

Fatigue Index

No group × time interaction effect was observed for fatigue index (F = 0.04, $p = 0.85$, $\eta_p^2 = 0.002$). However, fatigue index significantly increased from pre- to post-experimental intervention (F = 9.57, $p = 0.01$, $\eta_p^2 = 0.374$). There were no differences between groups (F = 0.23, $p = 0.64$, $\eta_p^2 = 0.014$).

Total Work

There were no differences between groups (F = 0.11, $p = 0.75$, $\eta_p^2 = 0.007$) and no group × time interaction effect for total work (F = 0.12, $p = 0.74$, $\eta_p^2 = 0.007$). However, total work significantly increased after the experimental intervention (F = 14.57, $p = 0.002$, $\eta_p^2 = 0.477$). All WaNT testing results are shown in Table 9.

Resistance Training

Muscular Strength and Training Volume

There was no group × time interaction effect (F = 0.88, $p = 0.36$, $\eta_p^2 = 0.052$) for back squat 1-RM and no differences between groups (SUPP: 145.58 ± 27.42 kg vs. NONSUPP: 150.38 ± 39.17 kg, F = 0.09, $p = 0.76$, $\eta_p^2 = 0.006$). Back squat 1-RM

significantly increased from pre- to post-intervention (Pre-1-RM: 139.14 ± 32.88 kg vs. Post 1-RM: 156.82 ± 32.43 kg, F = 53.26, $p < 0.0001$, $\eta_p^2 = 0.769$). Regarding back squat training volume, there was a significant group × week interaction effect for back squat volume (F = 5.47, $p = 0.003$, $\eta_p^2 = 0.296$). The SUPP completed a significantly greater back squat volume during week 4 compared to week 1 ($p < 0.05$). The NONSUPP group did significantly more volume during weeks 2 and 4 compared to week 3 ($p < 0.05$). There were no differences between groups at any week ($p > 0.05$) (Figure 2).

No group × time interaction effect (F = 0.13, $p = 0.26$, $\eta_p^2 = 0.077$) was noted for bench press 1-RM. Bench press 1-RM was not significantly different between groups (SUPP: 109.47 ± 14.74 kg vs. NONSUPP: 112.75 ± 21.84 kg, F = 0.16, $p = 0.69$, $\eta_p^2 = 0.010$). Regarding time, bench press 1-RM significantly increased from pre- to post-intervention (Pre 1-RM: 105.18 ± 16.50 kg vs. Post 1-RM: 117.05 ± 18.79 kg, F = 20.61, $p < 0.0001$, $\eta_p^2 = 0.563$). There was no group × week interaction effect for bench press training volume (F = 0.22, $p = 0.88$, $\eta_p^2 = 0.017$), but a significant time effect (F = 6.75, $p = 0.001$, $\eta_p^2 = 0.342$). Post-hoc comparisons revealed significant greater bench press volume in week 2 compared to week 1 ($p = 0.02$) and a significant decrease in week three from week 2 ($p = 0.002$). There were no differences between groups (F = 0.40, $p = 0.54$, $\eta_p^2 = 0.029$). Bench press training volumes are shown in Figure 3.

There was no group × week interaction effect for pull-up volume (F = 2.81, $p = 0.08$, $\eta_p^2 = 0.178$). There were also no differences in pull-up volume across weeks (F = 0.61, $p = 0.56$, $\eta_p^2 = 0.045$) or between groups (F = 1. 7, $p = 0.32$, $\eta_p^2 = 0.076$). (Figure 4).

There was a group × week interaction effect for pushup volume (F = 3.16, p = 0.05, η_p^2 = 0.195). Pushup volume was significantly greater in weeks 3 and 4 compared to weeks 1 and 2 ($p < 0.05$). There were no differences in the number of pushups completed between weeks 1 and 2 or weeks 3 and 4 ($p > 0.05$). Pushup volume is presented in Figure 5.

Resistance Training Blood Glucose

There was no group × time × week interaction effect for exercise blood glucose (F = 1.10, p = 0.37, η_p^2 = 0.078). Blood glucose response during full body RT days did not differ between groups (F = 0.73, p = 0.41, η_p^2 = 0.053). However, there were significant main effects for time (F = 10.10, p = 0.001, η_p^2 = 0.437) and weeks (F = 4.22, p = 0.01, η_p^2 = 0.245). During RT sessions, blood glucose did not change from pre-exercise to mid-exercise (p = 0.08), but significantly increased from mid- to post-exercise (p = 0.01). Blood glucose responses during RT were significantly higher during week 3 than week 1 (p = 0.05), but there were no other week differences ($p > 0.05$). Blood glucose responses during RT sessions are presented in Table 10.

Peak Oxygen Consumption Test

Performance Markers

There was no group × time interaction effect for relative (F = 1.50, p = 0.24, η_p^2 = 0.086) or absolute (F = 2.14, p = 0.16, η_p^2 = 0.118) $\dot{V}O_{2\ peak}$. There were also no group differences with relative (SUPP: 43.66 ± 4.88 $ml \cdot kg^{-1} \cdot min^{-1}$ vs. NONSUPP: 44.55 ± 5.48 $ml \cdot kg^{-1} \cdot min^{-1}$, F = 0.18, p = 0.67, η_p^2 = 0.011) and absolute (SUPP: 3.79 ± 0.60 $L \cdot min^{-1}$ vs. NONSUPP: 3.76 ± 0.74 $L \cdot min^{-1}$, F = 0.01, p = 0.94, η_p^2 = 0.000) $\dot{V}O_{2\ peak}$. Regarding

time, participants significantly increased their relative (Pre $\dot{V}O_{2\ peak}$: 42.46 ± 5.12 ml·kg^{-1}·min^{-1} vs. Post $\dot{V}O_{2\ peak}$: 45.75 ± 4.72 ml·kg^{-1}·min^{-1}, F = 8.67, p = 0.01, η_p^2 = 0.351) and absolute (Pre $\dot{V}O_{2\ peak}$: 3.65 ± 0.66 L·min^{-1} vs. Post $\dot{V}O_{2\ peak}$: 3.90 ± 0.66 L·min^{-1}, F = 7.25, p = 0.02, η_p^2 = 0.312) $\dot{V}O_{2\ peak}$ from pre-to post-training. There was no group × time interaction effect for TTE (F = 0.22, p = 0.64, η_p^2 = 0.014). However, TTE significantly increased from pre-to post-training (Pre: 11.49 ± 2.38 min vs. Post $\dot{V}O_{2\ peak}$: 12.57 ± 2.44 min, F = 52.39, p < 0.0001, η_p^2 = 0.766), but there were no differences between groups (SUPP: 11.91 ± 2.45 min vs. NONSUPP: 12.15 ± 2.49 min, F = 0.04, p = 0.84, η_p^2 =0.040).

Cardiorespiratory Responses

With respect to HR, there was no group × time × stage interaction effect (F = 0.57, p = 0.50, η_p^2 = 0.042) (Figure 6). There were also no differences between groups (F = 0.006, p = 0.94, η_p^2 = 0.000) and time (F = 1.67, p = 0.22, η_p^2 = 0.114). However, there was a significant difference between stages (F = 79.60, p < 0.0001, η_p^2 = 0.860). HR significantly increased from during each stage from one to the end of the test (p < 0.05).

No group × time × stage interaction (F = 0.02, p = 0.98, η_p^2 = 0.001), group differences (F = 0.39, p = 0.54, η_p^2 = 0.029), or time differences (F = 0.51, p = 0.49, η_p^2 =0.037) were noted for breathing rate. Breathing rate significantly increased during each stage (F = 78.32, p < 0.0001, η_p^2 = 0.858). Regarding $\dot{V}E$, there was no group × time × stage interaction effect (F = 0.29, p = 0.64, η_p^2 = 0.022) and no differences between groups (F = 0.59, p = 0.46, η_p^2 = 0.043) or time (F = 1.89, p = 0.19, η_p^2 = 0.127). $\dot{V}E$ significantly increased during each stage (F = 183.31, p < 0.0001, η_p^2 = 0.934).

With respect to RER, there was no group × time × stage interaction effect ($F = 2.25$, $p = 0.13$, $\eta_p^2 = 0.158$) (Figure 7) or differences between groups ($F = 0.14$, $p = 0.71$, $\eta_p^2 = 0.012$). However, RER was significantly lower after the intervention ($F = 5.17$, $p = 0.04$, $\eta_p^2 = 0.301$) and there was a significant difference between stages ($F = 97.85$, $p < 0.0001$, $\eta_p^2 = 0.891$). Stages 1-3 were all significantly different from each other, with RER increasing during each stage ($p < 0.05$).

Blood Lactate

There was no group × time × stage interaction effect ($F = 0.30$, $p = 0.74$, $\eta_p^2 = 0.029$). Blood lactate did not significantly differ between groups ($F = 0.27$, $p = 0.61$, $\eta_p^2 = 0.027$) or times ($F = 1.09$, $p = 0.32$, $\eta_p^2 = 0.098$). However, there was a significant difference in blood lactate across stages ($F = 57.20$, $p < 0.0001$, $\eta_p^2 = 0.851$). Blood lactate did not differ from Stages 1 and 2 ($p = 0.6$), but significantly increased during each stage from Stage 2 to until the end of the test ($p < 0.05$). Blood lactate responses across groups, time, and stages are presented in Figure 8.

Rating of Perceived Exertion

There were no group × time × stage interaction effect ($F = 0.09$, $p = 0.97$, $\eta_p^2 = 0.018$) or differences in RPE for group ($F = 2.93$, $p = 0.11$, $\eta_p^2 = 0.163$) or time ($F = 0.27$, $p = 0.74$, $\eta_p^2 = 0.054$) (Figure 9). Regarding stage, RPE significantly differed between stages ($F = 281.39$, $p < 0.0001$, $\eta_p^2 = 0.949$). RPE significantly increased from each Stage 1 to the following stage until the end of the test ($p < 0.0001$).

Resting Blood Markers

Glucose

There was no group × time interaction effect (F = 0.18, $p = 0.68$, $\eta_p^2 = 0.012$) for glucose. There were also no differences in resting glucose between groups (SUPP: 84.05 ± 7.08 mg/dL vs. NONSUPP: 79.02 ± 7.62 mg/dL, F = 3.31, $p = 0.09$, $\eta_p^2 = 0.181$) or time (Pre-intervention: 79.9 ± 8.1 mg/dL vs. Post-intervention: 82.9 ± 6.9 mg/dL, F = 1.81, $p = 0.20$, $\eta_p^2 = 0.107$).

Testosterone

No group × time interaction effect (F = 0.02, $p = 0.66$, $\eta_p^2 = 0.013$) was noted for testosterone. Testosterone levels were not different between groups (SUPP: 5.04 ± 1.45 ng/ml vs. NONSUPP: 5.71 ± 1.94 ng/ml, F = 0.74, $p = 0.40$, $\eta_p^2 = 0.047$). There were no differences is testosterone levels from pre-to post experimental intervention (Pre-intervention: 5.54 ± 1.80 ng/ml vs. Post-intervention: 5.25 ± 1.71 ng/ml, F = 0.56, $p = 0.46$, $\eta_p^2 = 0.036$).

Insulin

There was no group × time interaction effect (F = 0.83, $p = 0.38$, $\eta_p^2 = 0.060$) for insulin. There was also no group (SUPP: 3.59 ± 3.45 mIU/L vs. NONSUPP: 2.75 ± 1.36 mIU/L, F = 0.43, $p = 0.53$, $\eta_p^2 = 0.032$) or time differences (Pre-intervention: 3.10 ± 2.76 mIU/L vs. Post-intervention: 3.21 ± 2.56 mIU/L, F = 0.10, $p = 0.75$, $\eta_p^2 = 0.008$) for insulin.

Gastrointestinal Distress

Nausea

No exercise × group × time × week interaction effect (F = 1.35, $p = 0.28$, $\eta_p^2 = 0.088$) was noted for nausea. No exercise (F = 0.23, $p = 0.64$, $\eta_p^2 = 0.016$), group (F = 0.18, $p = 0.68$, $\eta_p^2 = 0.012$) or week (F = 2.23, $p = 0.09$, $\eta_p^2 = 0.140$) differences were noted for nausea. However, nausea significantly increased from pre-to post-exercise (F = 26.14, $p < 0.0001$, $\eta_p^2 = 0.651$).

Regurgitation/Reflux

There was no exercise × group × time × week interaction effect (F = 1.64, $p = 0.21$, $\eta_p^2 = 0.105$) for regurgitation/reflux. Regurgitation/reflux significantly increased from pre- to post-exercise (F = 18.55, $p = 0.001$, $\eta_p^2 = 0.570$). There were no differences between exercises (F = 0.04, $p = 0.85$, $\eta_p^2 = 0.003$) or weeks (F = 2.23, $p = 0.135$, $\eta_p^2 = 0.137$).

Stomach Fullness

There was an exercise × group × time × week interaction effect (F = 3.77, $p = 0.02$, $\eta_p^2 = 0.225$). The SUPP group had significantly lower stomach fullness ($p = 0.02$) post-RT exercise than post HIIT exercise in week 2. The SUPP group also has significantly lower stomach fullness post-RT exercise in week 1 ($p = 0.02$) and week 3 ($p = 0.002$). There was no group (F = 0.83, $p = 0.38$, $\eta_p^2 = 0.060$), time (F = 4.21, $p = 0.06$, $\eta_p^2 = 0.244$), or exercise (F = 0.09, $p = 0.87$, $\eta_p^2 = 0.773$) differences for stomach fullness. Regarding week, there was a significant main effect (F = 4.87, $p = 0.02$, $\eta_p^2 = 0.272$). There was a significantly greater feeling of stomach fullness during week 1 than week 2

($p = 0.05$). Stomach fullness did not differ between weeks 2 and 4 or between weeks 1, 3, and 4 ($p > 0.05$).

Abdominal Cramps

There was no exercise × group × time × week interaction effect (F = 0.05, $p = 0.96$, $\eta_p^2 = 0.004$) for abdominal cramps. There were no differences in abdominal cramps between exercises (F = 2.66, $p = 0.13$, $\eta_p^2 = 0.159$), groups (F = 3.70, $p = 0.08$, $\eta_p^2 = 0.209$), or weeks (F = 0.29, $p = 0.70$, $\eta_p^2 = 0.020$). However, abdominal cramps increased from pre-to post-exercise (F = 15.86, $p = 0.001$, $\eta_p^2 = 0.53$).

Gas/Flatulence

There was no time (F = 3.80, $p = 0.07$, $\eta_p^2 = 0.221$), exercise (F = 3.27, $p = 0.58$, $\eta_p^2 = 0.023$), or week (F = 0.41, $p = 0.62$, $\eta_p^2 = 0.028$) differences for gas/flatulence and no exercise × group × time × week interaction effect (F = 0.37, $p = 0.70$, $\eta_p^2 = 0.026$). Regarding groups, there were no differences in gas/flatulence (F = 0.03, $p = 0.87$, $\eta_p^2 = 0.002$).

Urge to Defecate

There was no exercise × group × time × week interaction effect (F = 0.04, $p = 0.98$, $\eta_p^2 = 0.03$) for urge to defecate. There were no significant differences regarding groups (F = 0.04, $p = 0.84$, $\eta_p^2 = 0.003$), exercises (F = 0.79, $p = 0.39$, $\eta_p^2 = 0.053$), times (F = 3.22, $p = 0.94$, $\eta_p^2 = 0.187$), or weeks (F = 0.53, $p = 0.55$, $\eta_p^2 = 0.037$) for urge to defecate. All GID results are presented in Table 11.

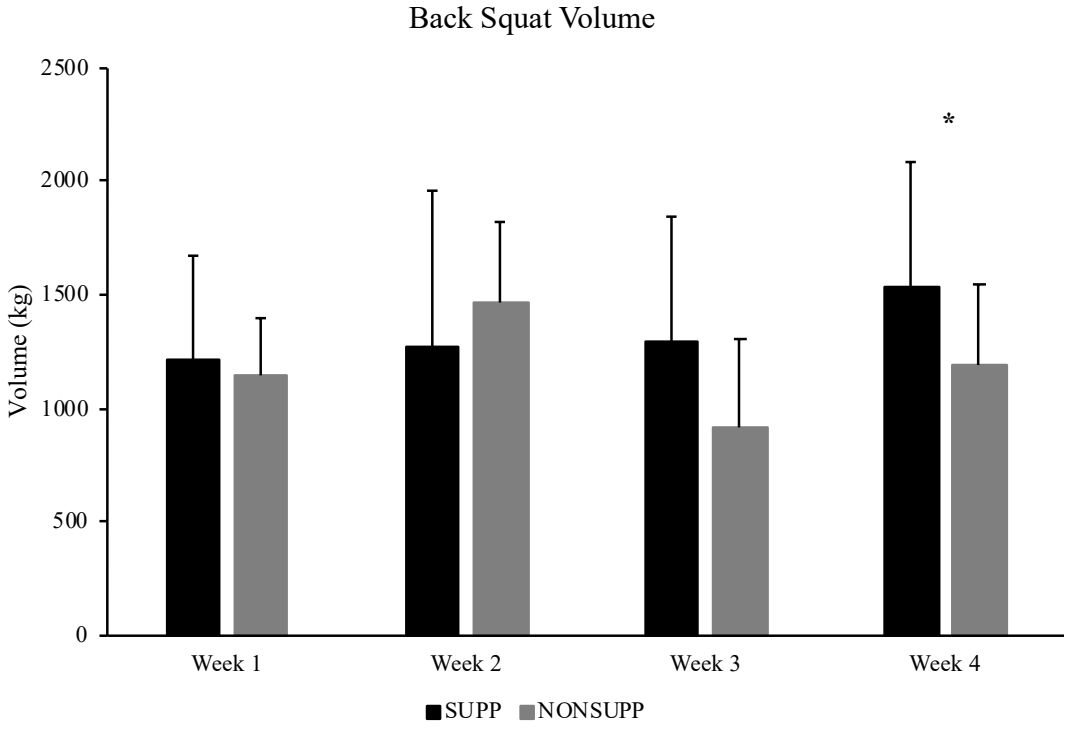

Figure 2. Back Squat Volume. Data are presented across the 4-week intervention as mean ± standard deviation for both supplemented (SUPP) and non-supplemented (NONSUPP) groups. *Significantly different from week 1 ($p < 0.05$). There were no differences between groups but there was a significant interaction effect. The SUPP completed a significantly greater back squat volume during week 4 compared to week 1 ($p < 0.05$). The NONSUPP group did significantly more volume during weeks 2 and 4 compared to week 3 ($p < 0.05$).

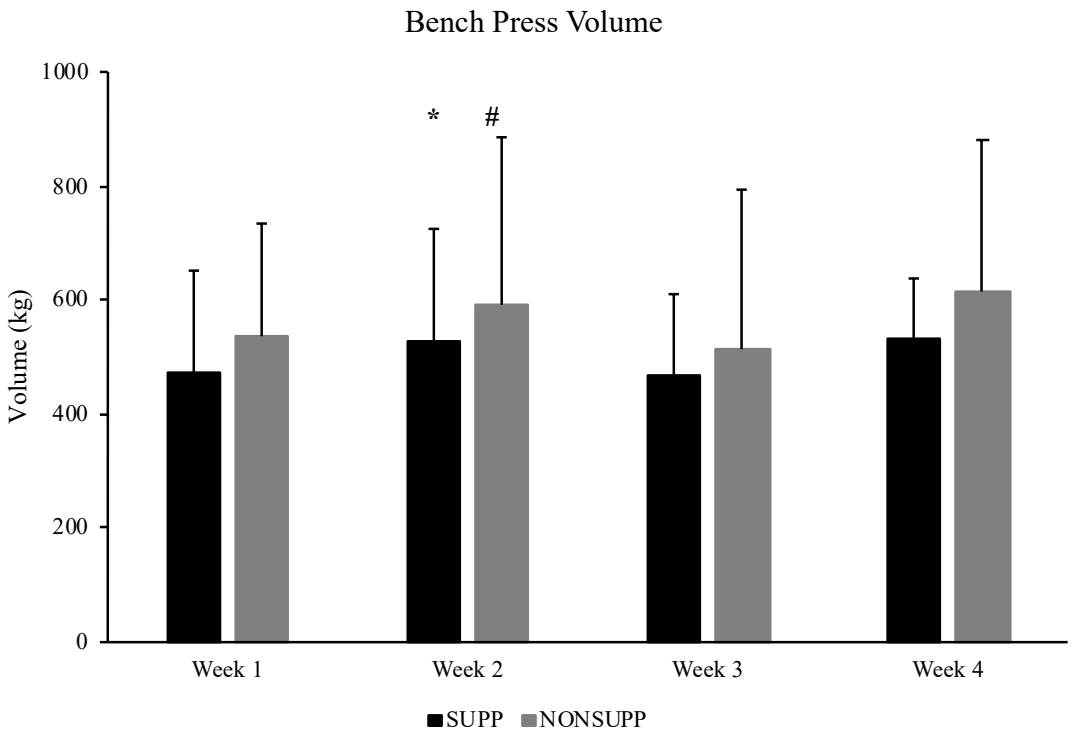

Figure 3. Bench press volume. Data are presented across the 4-week intervention as mean ± standard deviation for both supplemented (SUPP) and non-supplemented (NONSUPP) groups. *Significantly different from week 1 ($p < 0.05$). #Significantly different from week 3 ($p < 0.05$). There were no differences between groups and no interaction effect.

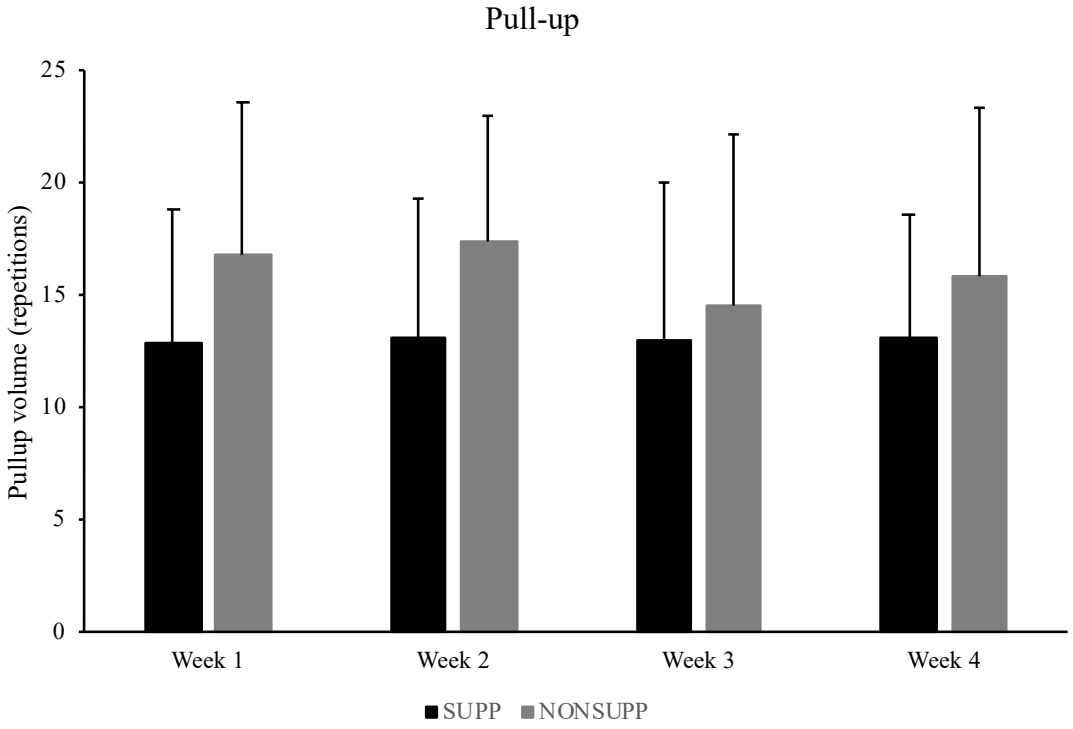

Figure 4. Pull-up Volume. Data are presented across the 4-week intervention as mean ± standard deviation for both supplemented (SUPP) and non-supplemented (NONSUPP) groups. There was no difference between weeks or groups and there was no interaction effect ($p > 0.05$).

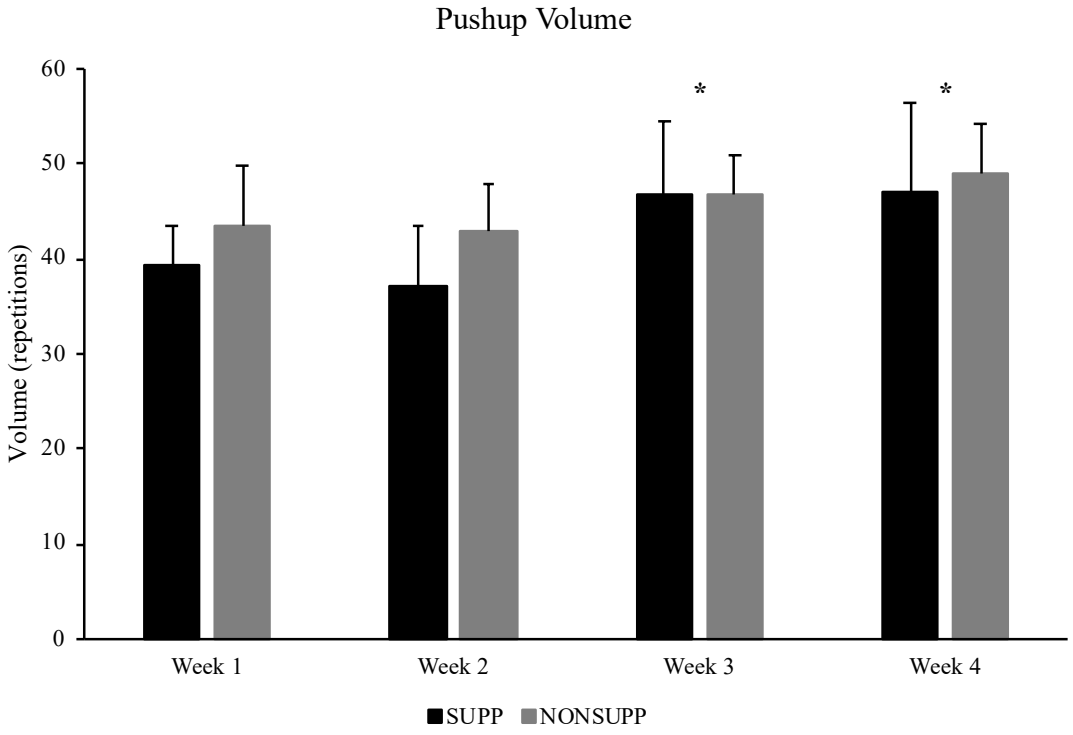

Figure 5. Pushup Volume. Data are presented across the 4-week intervention as mean ± standard deviation for both supplemented (SUPP) and non-supplemented (NONSUPP) groups. There was no difference between groups and there was no interaction effect. *Significantly different from weeks 1 and 2 ($p < 0.05$).

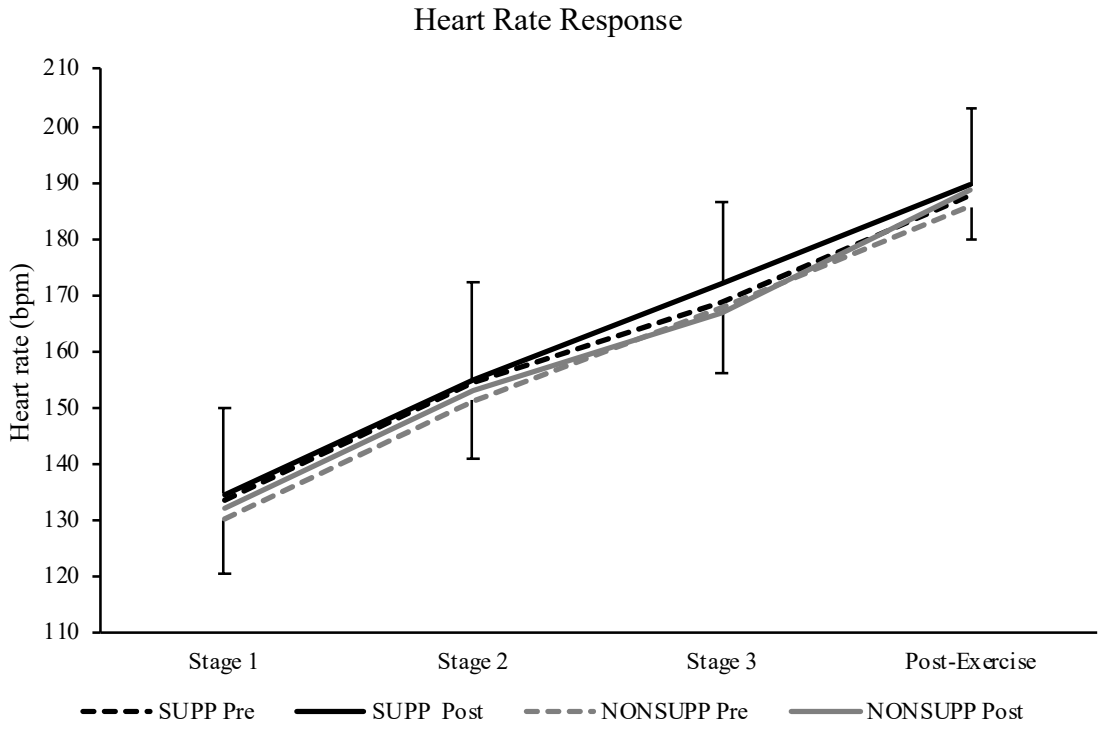

Figure 6. Heart Rate Responses during the $\dot{V}O_{2\ peak}$ Test. Data represent heart rate response across the first three stages and post-exercise of the $\dot{V}O_{2\ peak}$ test for both pre- and post-experimental intervention in the supplemented (SUPP) and non-supplemented (NONSUPP) groups. There were no differences between groups, time, and no interaction effect. Heart rate significantly increased from each stage until the end of the tests.

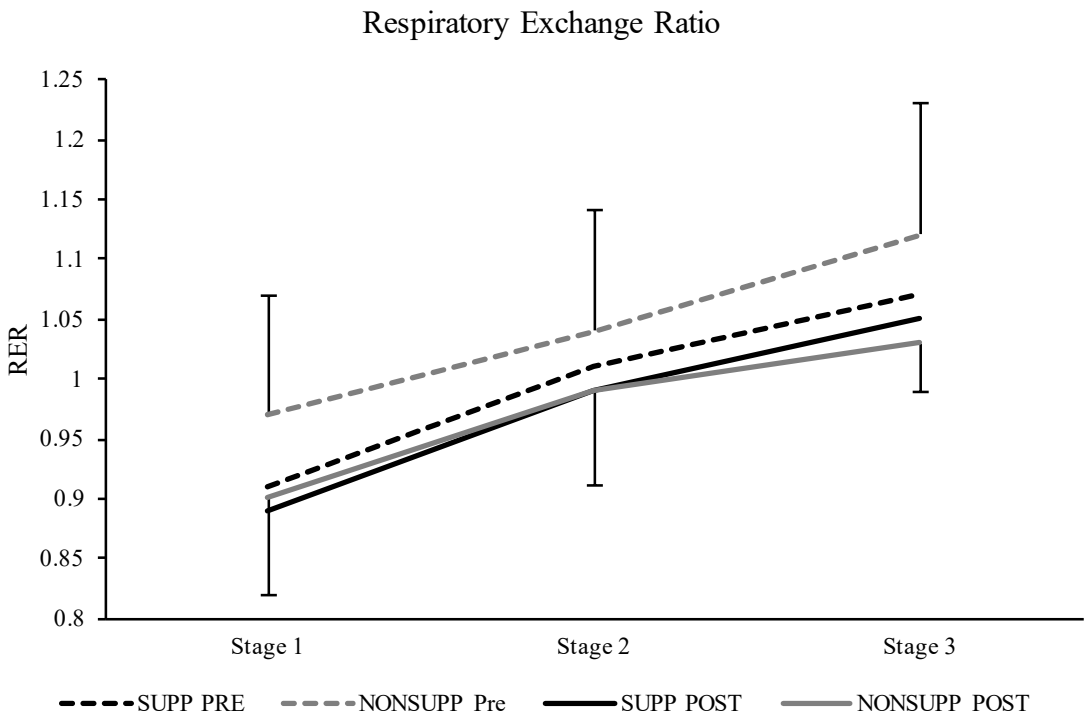

Figure 7. Respiratory Exchange Ratio during the $\dot{V}O_{2\ peak}$ Test. Data represent respiratory exchange ratio across the first three stage of the $\dot{V}O_{2\ peak}$ test both pre-and post-experimental intervention in the supplemented (SUPP) and non-supplemented (NONSUPP) groups. There were no differences between groups and no interaction effect. Respiratory exchanged ratio, averaged across the three stages, was significantly lower ($p = 0.02$) during the post-experimental intervention. Respiratory exchange ratio also significantly increased during each stage ($p < 0.05$).

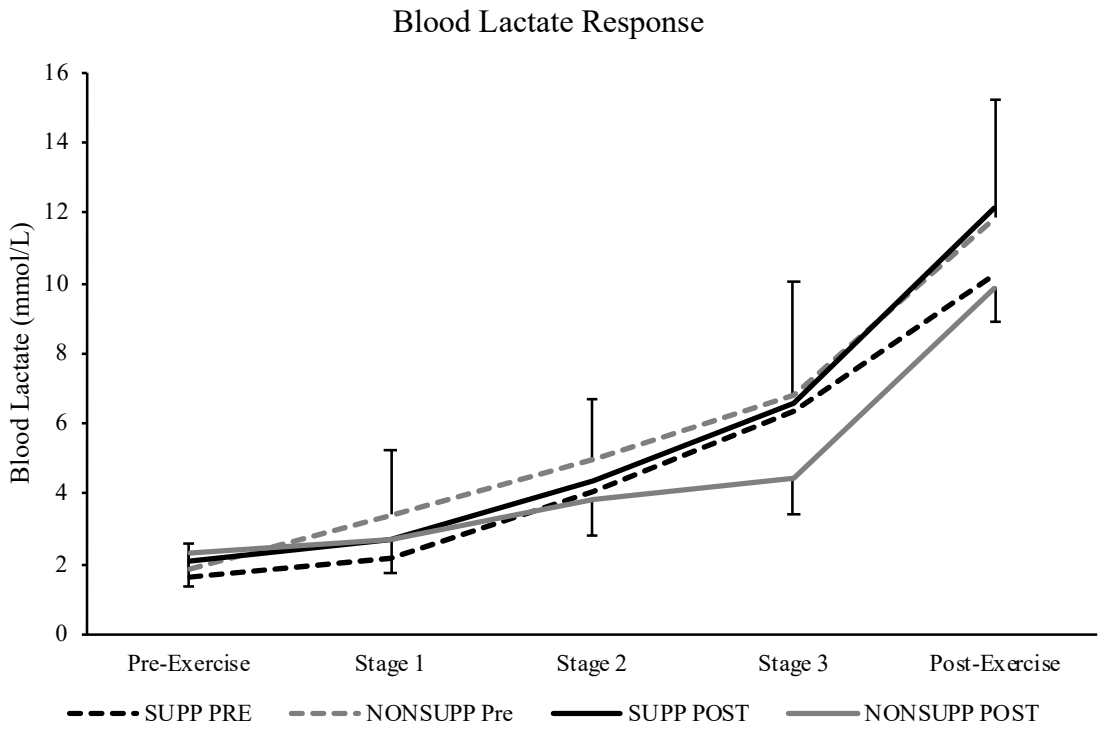

Figure 8. Blood Lactate Responses during the $\dot{V}O_{2\ peak}$ Test. Data represent blood lactate responses pre-exercise, during the first three stage of the $\dot{V}O_{2\ peak}$ test, and post-exercise both pre-and post-experimental intervention in the supplemented (SUPP) and non-supplemented (NONSUPP) groups. There were no differences between groups or time and no interaction effect. Blood lactate ($p < 0.05$) significantly increased from stage 2 until the end of the test.

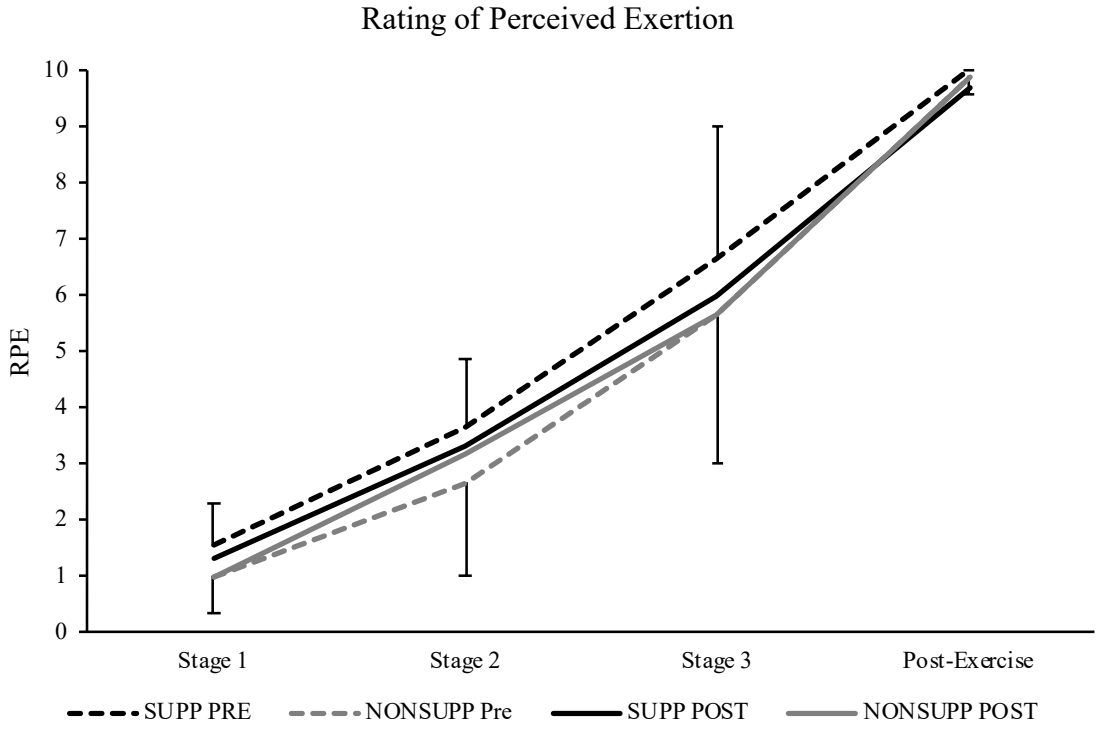

Figure 9. Rating of Perceived Exertion during the $\dot{V}O_{2\ peak}$ Test. Data represent rating of perceived exertion across the first three stages and post-exercise of the $\dot{V}O_{2\ peak}$ test both pre-and post-experimental intervention in the supplemented (SUPP) and non-supplemented (NONSUPP) groups. There were no differences between groups or time and no interaction effect. Rating of perceived exertion significantly increased during each stage until the end of the test ($p < 0.05$).

Table 3

Baseline Characteristics

Characteristic	SUPP (*n* = 9)	NONSUPP (*n* = 9)	*P* - value
Age (years)	21.0 ± 1.93	19.67 ± 1.0	0.09
Height (cm)	179.21 ± 7.05	179.32 ± 6.37	0.97
Body Mass (kg)	86.71 ± 9.62	83.80 ± 12.62	0.59
Body Fat (%)	19.44 ± 4.75	14.58 ± 6.08	0.08
Fat Mass (kg)	16.91 ± 4.45	12.35 ± 5.60	0.07
Fat Free Mass (kg)	69.79 ± 8.13	71.45 ± 11.37	0.73
Peak Power (Watts)	1152.89 ± 179.62	1137.11 ± 254.62	0.88
Mean Power (Watts)	706.11 ± 97.21	726.0 ± 122.43	0.71
Absolute $\dot{V}O_{2\ peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	3.73 ± 0.65	3.57 ± 0.69	0.61
Relative $\dot{V}O_{2\ peak}$ ($L \cdot min^{-1}$)	42.71 ± 5.42	42.23 ± 5.13	0.85
Back Squat 1-RM (kg)	135.61 ± 27.74	142.68 ± 38.73	0.66
Bench Press 1-RM (kg)	105.05 ± 12.85	105.30 ± 20.33	0.98

Data are presented as mean ± standard deviation. SUPP= supplemented group, NONSUPP= non-supplemented group.

Table 4

Anthropometrics

Characteristic	Week 1	Week 2	Week 3	Week 4
Body Mass (kg)				
SUPP	86.46 ± 9.52	86.26 ± 9.33	86.23 ± 9.39	86.24 ± 9.41
NONSUPP	84.32 ± 12.56	84.21 ± 12.70	84.31 ± 13.23	84.40 ± 13.34
	Baseline	**Post-Intervention**		
Body Fat (%)				
SUPP	19.44 ± 4.75	18.61 ± 4.02		
NONSUPP	14.58 ± 6.08	14.21 ± 5.05		
Fat Mass (kg)				
SUPP	16.91 ± 4.45	15.99 ± 3.64		
NONSUPP	12.35 ± 5.60	12.21 ± 4.95		
Fat Free Mass (kg)				
SUPP	69.79 ± 8.13	70.04 ± 8.08		
NONSUPP	71.45 ± 11.37	72.15 ± 11.22		

Data are presented as mean ± standard deviation. Body mass is reported across the 4-week experimental intervention. Body fat percentage, fat mass, and fat free mass are reported pre- and post-experimental intervention. SUPP= supplemented group, NONSUPP= non-supplemented group.

Table 5

Ultrasonography

Muscle	Pre-Intervention	Post-Intervention
Biceps (cm)	4.36 ± 0.50	4.54 ± 0.45*
CV (%)	1.94	1.21
Triceps (cm)	4.14 ± 0.71	4.24 ± 0.81
CV (%)	2.40	1.26
Gastrocnemius (cm)	2.22 ± 0.23	2.27 ± 0.45
CV (%)	1.16	1.27
Quadriceps (cm)	4.23 ± 0.68	3.99 ± 0.70
CV (%)	1.91	0.80
Hamstrings (cm)	3.78 ± 0.71	3.65 ± 0.52
CV (%)	2.13	1.30

Data are presented as mean ± standard deviation. Average muscle thickness of both groups combined for each muscle is reported for pre- and post-experimental intervention. *Significantly greater than pre-intervention ($p < 0.05$). CV= coefficient of variation.

Table 6

Dietary Carbohydrate Intake

		28-day Low Carbohydrate, High Fat Diet			
	Habitual Diet	**Week 1**	**Week 2**	**Week 3**	**Week 4**
CHO (E%)					
SUPP	43.16 ± 7.70*	25.33 ± 4.09	24.58 ± 2.88	23.86 ± 2.15	23.16 ± 2.80
NONSUPP	44.21 ± 5.18*	22.94 ± 4.87	21.94 ± 4.43	22.62 ± 2.03	23.33 ± 3.43
CHO (g)					
SUPP	251.14 ± 84.50*	174.89 ± 26.60	174.13 ± 20.40	159.08 ± 27.87	171.55 ± 34.21
NONSUPP	298.71 ± 51.03*	169.51 ± 29.29	159.38 ± 39.36	160.11 ± 15.90	176.84 ± 35.71
CHO (g/kg)					
SUPP	2.98 ± 1.28*	2.02 ± 0.20	2.02 ± 0.13	1.85 ± 0.29	1.98 ± 0.24
NONSUPP	3.66 ± 0.93*	2.05 ± 0.51	1.87 ± 0.24	1.92 ± 0.22	2.09 ± 0.19

Data are presented as mean ± standard deviation. Carbohydrate intakes are reported for the habitual diet and across the 4-week experimental intervention. *Significantly greater than weeks 1-4 ($p < 0.05$). SUPP= supplemented group, NONSUPP= non-supplemented group.

Table 7

Dietary Protein Intake

		28-day Low Carbohydrate, High Fat Diet			
	Habitual Diet	**Week 1**	**Week 2**	**Week 3**	**Week 4**
Protein (E%)					
SUPP	23.94 ± 5.44	25.84 ± 3.14	25.47 ± 1.89	24.88 ± 1.87	24.82 ± 2.60
NONSUPP	22.41 ± 9.01	27.43 ± 2.72	26.45 ± 3.79	25.02 ± 1.54	25.06 ± 2.31
Protein (g)					
SUPP	134.10 ± 27.28*	178.19 ± 18.80	180.82 ± 18.99	173.20 ± 23.58	184.52 ± 27.66
NONSUPP	124.86 ± 35.60*	205.59 ± 30.28	189.63 ± 21.36	177.16 ± 15.42	190.42 ± 32.35
Protein (g/kg)					
SUPP	1.58 ± 0.42*	2.07 ± 0.16	2.11 ± 0.25	2.02 ± 0.24	2.14 ± 0.19
NONSUPP	1.51 ± 0.42*	2.52 ± 0.72	2.30 ± 0.47	2.13 ± 0.27	2.30 ± 0.53

Data are presented as mean ± standard deviation. Protein intakes are reported for the habitual diet and across the 4-week experimental intervention. *Significantly lower than weeks 1-4 ($p < 0.05$). Protein intake (g/kg) was significantly higher for the NONSUPP group overall ($p < 0.05$). SUPP= supplemented group, NONSUPP= non-supplemented group.

Table 8

Dietary Fat Intake

		28-day Low Carbohydrate, High Fat Diet			
	Habitual Diet	**Week 1**	**Week 2**	**Week 3**	**Week 4**
Fat (E%)					
SUPP	30.43 ± 5.82*	48.22 ± 5.96	49.98 ± 4.21	49.41 ± 3.80	51.29 ± 4.68
NONSUPP	32.43 ± 7.84*	49.63 ± 5.71	51.62 ± 4.57	52.37 ± 2.88	51.62 ± 3.98
Fat (g)					
SUPP	78.22 ± 26.12*	149.89 ± 27.47	158.90 ± 24.20	158.16 ± 12.17	169.52 ± 29.49
NONSUPP	101.14 ± 16.12*	166.46 ± 38.80	167.04 ± 33.22	166.78 ± 24.31	175.74 ± 38.64
Fat (g/kg)					
SUPP	0.92 ± 0.37*	1.77 ± 0.48	1.87 ± 0.40	1.85 ± 0.27	1.99 ± 0.46
NONSUPP	1.25 ± 0.34*	2.00 ± 0.51	2.01 ± 0.44	1.98 ± 0.28	2.12 ± 0.57

Data are presented as mean ± standard deviation. Fat intakes are reported for the habitual diet and across the 4-week experimental intervention. *Significantly lower than weeks 1-4 ($p < 0.05$). Fat intake (g/kg and g) was significantly higher for the NONSUPP group overall ($p < 0.05$). SUPP= supplemented group, NONSUPP= non-supplemented group.

Table 9

Wingate Testing

Variable	Pre-Intervention	Post-Intervention
Peak Power (watts)		
SUPP	1152.89 ± 179.62	1258.11 ± 165.03
NONSUPP	1137.11 ± 254.62	1241.11 ± 257.02
Mean Power (watts)		
SUPP	706.11 ± 97.21	744.67 ± 81.88*
NONSUPP	726.00 ± 122.43	757.89 ± 124.19*
Total Work (joules)		
SUPP	21182.83 ± 2918.86	22338.40 ± 2451.36*
NONSUPP	21770.21 ± 3660.55	22735.18 ± 3730.49*
Fatigue Index (watts/second)		
SUPP	25.60 ± 4.37	28.86 ± 4.86*
NONSUPP	24.61 ± 6.83	27.49 ± 6.25*
Relative Mean Power (watts/kg)		
SUPP	8.18 ± 0.53	8.69 ± 0.53*
NONSUPP	8.64 ± 0.53	8.91 ± 0.68*
Relative Peak Power (watts/kg)		
SUPP	13.33 ± 1.07	14.66 ± 1.18*
NONSUPP	13.39 ± 1.25	14.47 ± 0.95*

Data are presented as mean ± standard deviation. Wingate testing results are reported for pre-and post-experimental intervention for both groups. *Significantly greater than pre-intervention ($p < 0.05$). SUPP= supplemented group, NONSUPP= non-supplemented group.

Table 10

Resistance Training Blood Glucose

Time	Week 1	Week 2	Week 3	Week 4
Pre-Exercise				
SUPP	80.75 ± 9.32	79.33 ± 7.25	88.25 ± 7.48*	85.78 ± 6.78
NONSUPP	85.44 ± 6.35	84.56 ± 8.83	89.38 ± 7.60*	88.11 ± 11.14
Mid-Exercise				
SUPP	93.50 ± 18.60	95.67 ± 13.02	104.13 ± 15.59*	94.56 ± 9.49
NONSUPP	88.67 ± 11.74	91.56 ± 10.62	91.75 ± 15.44*	91.67 ± 9.35
Post-Exercise				
SUPP	98.88 ± 14.15	99.22 ± 18.01	108.00 ± 21.51*	103.89 ± 16.10
NONSUPP	90.22 ± 14.95	99.00 ± 13.67	99.00 ± 18.48*	98.22 ± 10.40

Data are presented as mean ± standard deviation. Blood glucose values are presented in mg/dL and represent changes between groups, time, and weeks. *Significantly greater than week 1 ($p < 0.05$). Blood glucose significantly increased from mid- to post-exercise ($p < 0.05$). SUPP= supplemented group, NONSUPP= non-supplemented group.

Table 11

Gastrointestinal Distress

Variable	Week 1	Week 2	Week 3	Week 4
Nausea				
Pre-Exercise				
SUPP	0.61 ± 1.09	0.11 ± 0.47	0.28 ± 0.57	0.06 ± 0.24
NONSUPP	0.12 ± 0.33	0.22 ± 0.55	0.56 ± 0.92	0.29 ± 0.69
Post-Exercise				
SUPP	1.78 ± 2.80	1.17 ± 2.23	1.22 ± 2.29	0.72 ± 2.19
NONSUPP	2.71 ± 1.86	1.78 ± 2.29	1.11 ± 1.81	1.53 ± 2.27
Regurgitation/ Reflux				
Pre-Exercise				
SUPP	0.39 ± 0.98	0.28 ± 0.75	0.44 ± 0.98	0.06 ± 0.24
NONSUPP	0.24 ± 0.44	0.50 ± 1.04	0.28 ± 0.57	0.29 ± 0.69
Post-Exercise				
SUPP	1.78 ± 2.56	1.00 ± 2.09	0.72 ± 1.49	0.72 ± 2.14
NONSUPP	2.47 ± 1.84	1.17 ± 1.76	1.11 ± 1.91	1.12 ± 1.90
Stomach Fullness				
Pre-Exercise				
SUPP	3.39 ± 2.83	2.06 ± 2.36	2.17 ± 2.98	2.17 ± 3.01
NONSUPP	3.41 ± 2.15	2.06 ± 1.95	1.33 ± 1.57	1.41 ± 1.70
Post-Exercise				
SUPP	3.11 ± 2.65	2.72 ± 3.14	2.89 ± 3.66	2.06 ± 2.65
NONSUPP	4.41 ± 2.94	2.67 ± 2.81	2.06 ± 2.18	2.24 ± 2.66
Abdominal Cramps				
Pre-Exercise				
SUPP	0.06 ± 0.24	0.33 ± 1.19	0.11 ± 0.47	0.00 ± 0.00
NONSUPP	0.00 ± 0.00	0.00 ± 0.00	0.11 ± 0.47	0.24 ± 0.66
Post-Exercise				
SUPP	1.06 ± 1.80	1.17 ± 1.79	0.67 ± 1.08	0.44 ± 0.78
NONSUPP	0.88 ± 2.00	1.17 ± 1.76	1.11 ± 1.91	1.12 ± 1.90
Gas/ Flatulence				
Pre-Exercise				
SUPP	0.94 ± 2.04	0.83 ± 1.58	0.67 ± 1.33	0.67 ± 1.37
NONSUPP	0.53 ± 0.80	0.33 ± 0.49	0.39 ± 0.61	0.29 ± 0.59
Post-Exercise				
SUPP	1.28 ± 2.05	0.94 ± 1.95	0.94 ± 2.18	0.89 ± 2.17
NONSUPP	1.00 ± 1.50	0.28 ± 0.57	0.67 ± 1.33	0.76 ± 1.30
Urge to Defecate				
Pre-Exercise				
SUPP	1.67 ± 2.59	0.83 ± 1.29	1.11 ± 1.75	1.11 ± 1.94
NONSUPP	0.53 ± 1.01	0.28 ± 0.46	0.39 ± 0.70	0.47 ± 1.07
Post-Exercise				
SUPP	1.56 ± 2.23	1.28 ± 2.54	1.28 ± 2.78	1.78 ± 2.94
NONSUPP	0.53 ± 1.33	0.17 ± 0.51	0.61 ± 1.20	0.76 ± 1.25

Data are presented as mean ± standard deviation. Gastrointestinal distress is reported on a 1-10 likert scale. SUPP= supplemented group; NONSUPP= non-supplemented group.

CHAPTER V

DISCUSSION

The purpose of this investigation was to examine the effects of CHO timing while consuming a LCHF diet and participating in a high-intensity exercise program. Overall, the results from this study suggest that 4 weeks of concurrent RT and HIIT and consuming a LCHF diet significantly increases muscular strength, muscular endurance, muscular thickness, power output, and aerobic metabolism, but does not improve resting blood biomarkers or body composition. However, there were no differences between treatment groups suggesting CHO timing has little to no effect on adaptations to high-intensity exercise. Other positive training/diet outcomes from the current investigation include significant reductions in RER and an increase in TTE during the $\dot{V}O_{2\ peak}$ test. Regarding GID, there were no differences between RT of HIIT on inducing GID symptoms, but there was a significant increase in GID for three of the six reported symptoms from pre-to post-exercise independent of treatment groups.

LCHF diets have been of recent interest due to the reported effects on metabolism, endurance performance, and health markers (Burke et al., 2002; Burke et al., 2017; Goedecke et al., 1999; Heatherly et al., 2017; McSwiney et al., 2017; Rowlands & Hopkins, 2002; Volek et al., 2016; Zajac et al., 2014; Zinn, Wood, Williden, Chatterton, & Maunder, 2017). Many have hypothesized that LCHF diets may be more advantageous for endurance athletes, due to increases in the ability to utilize fat (Noakes, Volek, &

Phinney, 2014; Volek, Noakes, & Phinney, 2015). However, recent literature suggests that endurance athletes benefit more from high CHO diets and that LCHF diets reduce exercise economy (Burke et al., 2017). Moreover, the main purpose of this study was to focus on a resistance training population compared to an endurance population and a non-ketogenic LCHF diet (~50% daily kcal intake coming from dietary fat).

Recent evidence has shown that a LCHF diet (~67% of total energy) significantly reduces CHO metabolism during high-intensity cycling (Stellingwerff et al., 2006). In the present study, all participants followed a LCHF, with one group receiving 30 g of CHO during exercise and 40 g of CHO after exercise. We hypothesized that this strategy would allow the SUPP group to maintain/increase CHO metabolism during exercise sessions (Cox et al., 2010), allowing them to increase training effort. Also, ingesting 40 g of CHO immediately following exercise would replenish muscle glycogen stores by taking advantage of the initial glycogen resynthesis phase (Jentjens & Jeukendrup, 2003). However, without direct measurement of muscle glycogen, we cannot infer if training 5 days per week, consuming a LCHF diet, or a combination of both caused any differences in muscle glycogen levels between groups. Due to both groups significantly increasing muscular and aerobic performance during the intervention, training effort and muscle glycogen status seem to be unaffected by timing of CHO intake.

Blood glucose was measured during each full body RT session to examine potential changes across the sessions. Similar to previous investigations, regardless of ingesting CHO or not, blood glucose levels significantly increased from pre- to post-RT (Haff et al., 1999; Smith et al., 2017b; Wax et al., 2013). CHO ingestion may have increased blood glucose levels in the SUPP group, but the glucogenic hormones cortisol

(Bird, Tarpenning, & Marino, 2006a, 2006c), growth hormone, and the catecholamines (epinephrine, norepinephrine) may have driven elevated blood glucose levels in the NONSUPP group. Therefore, both groups had significantly elevated blood glucose levels from pre- to post-exercise. In a classic study by McMillan et al. (1993), during a single RT session, without nutritional supplementation, it was observed that blood glucose significantly increased pre-to post-exercise coupled by an increase in epinephrine, norepinephrine, and growth hormone. Results from this investigation may explain the increases in the NONSUPP groups blood glucose levels, however, without measurement of glucogenic hormone concentrations around RT sessions, this is mere speculation.

One major argument for the efficacy of CHO ingestion during acute resistance exercise is that it can delay muscle glycogen degradation or replenish muscle glycogen during rest periods (Haff et al., 2003). Muscle glycogen has been shown to be significantly reduced following relative RT (Haff et al., 2000; Macdougall et al., 1999; Pascoe et al., 1993; Robergs et al., 1991; Tesch et al., 1986; Tesch et al., 1998) and reductions in muscle glycogen potentially lead to decreased performance (Leveritt & Abernethy, 1999). A major limitation of acute CHO supplementation during resistance exercise investigations (Haff et al., 2001; Haff et al., 1999; Krings et al., 2016; Lambert et al., 1991; Oliver et al., 2016; Rountree et al., 2017; Smith et al., 2017b; Wax et al., 2012; Wax et al., 2013; Wax et al., 2010) is the practicality of it being a viable nutrition strategy when practiced chronically. Our results suggest that over 20 training sessions (12 resistance training and 8 HIIT), 30 g of CHO ingested during each training session (~120 kcals) does not significantly influence RT or HIIT performance adaptations.

CHO supplementation has been shown to be efficacious during endurance exercise across a large number of studies (Stellingwerff & Cox, 2014). However, our training model was HIIT, which is different compared to typically studied aerobic exercise completed at ~55-70% $\dot{V}O_{2\ max}$ or $\dot{V}O_{2\ peak}$. During a typical HIIT session, the first 30 s sprint is primarily fueled by phosphocreatine and anaerobic glycolysis (Casey et al., 1996). Our study completed multiple bouts of maximal 30 s sprints (4-6 sprints) and after one sprint, the ensuing sprints become more reliant on aerobic metabolism (Bogdanis et al., 1996). Across the eight HIIT sessions, participants may have adapted to increasing lipid metabolism, which would in turn reduce reliance of CHO metabolism (Burgomaster et al., 2008). Even while consuming a LCHF, CHO metabolism from HIIT have not been enough to significantly reduce muscle glycogen from a training session.

Another potential explanation of no differences between CHO timing schedules could be due to the amount of energy expended during both RT and HIIT. Although RT session times were not recorded during each workout, the researchers noted that sessions typically lasted between 45-60 min depending on the RT day and 25.5-35 min for HIIT days. Based on estimations from RT sessions consisting of full body exercises and two sets of eight exercises (10 RM) net kcal expenditure is estimated at ~4.5 kcals/min (Stec & Rawson, 2012) and ~5 kcals/min (Rawson & Walsh, 2010). Based on these results, our participants would have been expending ~214-285 kcals per training session. Furthermore, energy expenditure during a similar HIIT protocol to the utilized in the present investigation observed an energy expenditure of ~175 kcals and ~7 kcals/min across four repeated sprints (Hazell, Olver, Hamilton, & Lemon, 2012). During the final 2 weeks of training participants progressed to five and six sprints, increasing energy

expenditure to ~214 kcals and ~245 kcals per session. With the SUPP group only receiving ~120 kcals of CHO during exercise, the relatively low amount of energy expenditure across RT and HIIT sessions was not enough to elicit a meaningful difference in performance from the NONSUPP group. Ingesting ~160 kcals of CHO after exercise was also not enough to alter muscle glycogen stores after exercise. Based on both of these conclusions, each participant came to training sessions with similar CHO fuel stores. Potential changes in CHO utilization and fuel stores may have been seen if we provided the SUPP group with a greater amount of CHO post-exercise (Roy & Tarnopolsky, 1998) and/or utilized a traditional aerobic training model (Koivisto et al., 1985).

One of the interesting findings with this investigation was the maintenance of body mass between both groups across the 4-week intervention. Investigations examining a similar population (resistance trained males) while completing RT and consuming a ketogenic diet have observed significant reductions in body mass following 6 weeks (Gregory et al., 2017), 10 weeks (Wilson et al., 2017), and 12 weeks (Kephart et al., 2018) of dieting and training. Additionally, our laboratory recently showed that 15 days on a similar LCHF to the present investigation resulted in a significant reduction in body mass (Waldman et al., 2017). Reductions in body mass during CHO restriction are most likely explained by reductions in muscle glycogen. Therefore, body mass changes may not be due to fat mass reductions but losses in whole body water. This remains as one of the limitations of LCHF research due to the lack of measurement of whole body water concentrations pre- and post-dietary/training interventions.

Due to the poor dietary habits of our participants and expected increased daily caloric expenditure from training 5 days per week, we increased caloric intake for 11 of the 18 participants. On average, participants increased daily kcal intake from their habitual diet by ~420 kcals. Increasing caloric intake may explain the maintenance of body mass across 4 weeks. With the increase in overall kcal intake, those kcals came from significantly increasing dietary fat and PRO intake while reducing CHO intake from the habitual diet. Other explanations for the maintenance of body mass may have been from gluconeogenesis converting amino acids to glucose and glucose maintaining muscle glycogen levels or RT males not requiring daily CHO intakes > 2 $g \cdot kg^{-1}$. The lower recommendations of CHO intake for RT males is an argument that has recently been suggested in the literature (Escobar et al., 2017).

Regarding other body composition variables, percentage body fat, fat mass, and fat free mass remained unchanged from pre- to post-experimental intervention. There was no significant difference in percentage body fat and fat mass between groups, although there were large differences ~5% and ~4.5 kg ($p = 0.07$) between both groups. However, the SUPP group had a significantly greater amount of fat pre- and post-intervention suggesting no changes due to the diet or training program. Furthermore it has been shown that percent body fat does not correlate to an individual's ability to burn fat and the authors do not see this as a limitation (Venables, Achten, & Jeukendrup, 2005). It was hypothesized that body composition, specifically fat free mass, would increase during the 4-week experimental intervention. Besides utilizing air displacement plethysmography (BodPod), which revealed no changes, the investigators decided to quantify fat free mass

with ultrasound. The ultrasound technique has been shown to be valid and reliable when measuring muscle thickness compared to MRI (Reeves, Maganaris, & Narici, 2004).

A significant increase in biceps thickness for both groups was observed, but muscle thickness remained unchanged for the triceps, gastrocnemius, quadriceps, and hamstrings. Previous research has shown that significant increases in muscle thickness can be seen in as little as 3 to 4 weeks (Seynnes et al., 2007; Stock et al., 2016), but are typically seen during longer training studies (Abe et al., 2000; Wilson et al., 2017). Increases in biceps thickness in the present investigation may be explained due to training of bent-over row, hammer curls, and upright rows during each upper body training sessions. However, since the four other muscles did significantly change as a result of the investigation it is difficult to interpret on seeing biceps increases. The short-duration of the investigation, training volume, and training status of our participants may partially explain no changes in triceps, gastrocnemius, hamstrings, and quadriceps. The 4-week experimental intervention utilized training loads of 75-85 % 1-RM corresponded to 6-8 reps for bilateral exercises and 4-5 per leg for unilateral exercises. We also implemented reps to failure training on full-body training sessions, to track training volume, but there were no meaningful differences between bench press volumes across weeks. However, there was a significant interaction for back squat training volume. The interaction revealed that the SUPP group did more back squat volume in week 4 compared to week 1 and the NONSUPP did more volume in weeks 2 and 4 compared to week 3. There were no significant differences between groups at week 4, but the SUPP group continued to increase back squat volume compared to the NONSUPP group from week 1. Although, not statistically significant potentially due to participant variation, the SUPP group was

potentially enhancing back squat training volume, suggesting CHO timing played in important role during week 4. Furthermore, during week 4, HIIT increased to six sprints, increasing overall lower body training demand during that week. However, additional research is needed to fully elucidate these inferences, specifically by increasing the length of our study. The training volumes in our study may have been too low to promote muscle hypertrophy of the larger muscle groups (Schoenfeld, Ogborn, & Krieger, 2017). Concurrently RT and HIIT for 4-weeks elicits an increase in biceps thickness measured via ultrasound but no significant differences in overall fat free mass measured via BodPod.

Although we employed a shorter duration training protocol we found significant and meaningful increases in bench press and back squat 1-RM in an already well-trained male. However, we only collected 1-RM bench press and back squat prior to the 2-week familiarization training and after the 4-week experimental intervention. Therefore, the increases in strength cannot be fully attributed to changes during the 4-week experimental intervention. Nonetheless, our RT protocol was effective in increasing upper body strength, lower body strength, and pushup performance. As previously stated, there were only increases in biceps thickness suggesting that strength increases may be explained through neural adaptations and/or muscle hypertrophy of musculature not measured. Our participants were intermediately trained according to the NSCA (Haff & Triplett, 2015), yet every participant was not familiar with each exercise, specifically the unilateral exercises. This may have led to enhanced neuromuscular adaptations, increasing muscular strength without structural changes. In order to minimize the proposed interference effect of concurrently training aerobic and strength training in the same

program (Hickson, 1980), the authors employed several training strategies. We utilized a HIIT model of aerobic exercise, cycled compared to running, and separated HIIT and RT days (Wilson et al., 2012). Moreover, it was recently shown that HIIT completed immediately after lower body RT for 16 sessions across two months did not inhibit increases in muscular strength and hypertrophy (Tsitkanou et al., 2017). Therefore, the observed RT adaptations were potentially maximized without the interference of HIIT.

Other low CHO diet investigations examining muscular strength have observed no changes (Kephart et al., 2018; Paoli et al., 2012; Van Zant et al., 2002) and increases in muscular strength (Wilson et al., 2017). However, Paoli et al. (2012) had subjects maintain their training regimen while consuming a low CHO diet and did not specify what the goal of their training program was. Van Zant et al. (2002) only utilized 4 weeks of training, and had subjects maintain training levels through treatment interventions, while not specifying training goals. Kephart et al. (2018) had subjects complete a CrossFit program for 12 weeks but did not specify the specific workouts completed. Similar to our study design, Wilson et al. (2017) utilized a periodized program, providing progression through the training program. A strength of our study, that may have also influenced strength adaptations, was the direct supervision of all participants during each training session. This ensured participants to maintain proper lifting form and may have enhance training effort (independent of CHO timing). Previous research has established that supervised training can produce superior strength outcomes compared to unsupervised training (Mazzetti et al., 2000).

We hypothesized that eight HIIT sessions would cause significant improvements in $\dot{V}O_{2\ peak}$. In agreement with our results, short duration HIIT investigations have shown

improvements in $\dot{V}O_{2\ peak}$ with only six HIIT sessions (Astorino et al., 2012; Hazell et al., 2010; Whyte et al., 2010). HIIT has become a popular training method for multiple populations (i.e. active adults, sedentary adults, athletes) due to its time efficiency and producing similar adaptations to traditional aerobic training (Burgomaster et al., 2008; Cochran et al., 2014; Gibala et al., 2006; Rakobowchuk et al., 2008). Burgomaster et al. (2005) showed that six sessions of HIIT did not significantly improvement $\dot{V}O_{2\ peak}$, but significantly improved oxidative enzyme capacity while increasing endurance capacity. Increases in absolute and relative $\dot{V}O_{2\ peak}$ may be explained by significant improvements at the cellular level. Specifically, participants may have become more efficient, due to increased levels of aerobic metabolism enzymes (i.e. citrate synthase, isocitrate dehydrogenase, cytochrome c oxidase). Furthermore, the percentage increases of relative (~7.7%) and absolute (~6.8%) $\dot{V}O_{2\ peak}$ from our data are similar to observations from Astorino et al. (2012) (relative (~5.5%) and absolute (6.5%). Regarding TTE, participants averaged ~1 min longer on the $\dot{V}O_{2\ peak}$ test coinciding with an increase in relative and absolute $\dot{V}O_{2\ peak}$. To ensure differences in TTE were not due to the learning effect with the cycle ergometer and headgear/mouthpiece/nose clip, we had participants complete three $\dot{V}O_{2\ peak}$ tests (one familiarization, pre-intervention, and post-intervention). Another explanation for our observed increases in $\dot{V}O_{2\ peak}$ were the poor aerobic fitness levels of our participants before beginning the study (Kaminsky, Arena, & Myers, 2015). We recruited participants who met the minimum ACSM physical activity guidelines (Medicine, 2013), but we were focused on their RT experience. Nonetheless, increasing aerobic fitness and muscular fitness are both positive adaptations for a healthy college aged male, especially over the course of 6 weeks.

Two other variables measured during the $\dot{V}O_{2\ peak}$ test included RER and blood lactate. It has been shown that consuming a LCHF diet decreases the ability to use CHO evident by reductions in pyruvate dehydrogenase activity and glycogenolysis (Stellingwerff et al., 2006). Blood lactate is product of glycolysis and increases in glycolytic flux can lead to increases in blood lactate, especially when lactate production is greater than lactate clearance (Brooks et al., 2005). Blood lactate significantly increased across the final two stages and at the end of the $\dot{V}O_{2\ peak}$ test. However, there were no differences between groups or time-points indirectly suggesting that CHO metabolism was maintained after the intervention. RER was also measured to analyze substrate oxidation. RER averaged across the first three stages of the $\dot{V}O_{2\ peak}$ test significantly decreased (1.02 vs. 0.98) from pre- to post-testing. Differences in RER may be attributed to utilizing more fat as a fuel source across the test (Burgomaster et al., 2008). Another explanation for a reduction in RER over the test is that participants became "fat-adapted" from consuming a LCHF diet for 4 weeks, which could be independent of training induced increases in fat metabolism. Recently Leckey et al. (2018) observed that increased consumption of dietary fats, rather than restriction of CHO, led to an increase in whole body fat oxidation. Without completion of a FAT_{max} Test (Randell et al., 2017), we are not able to infer if maximal fat oxidation ($g \cdot min^{-1}$) increased as a result of our study. However, our lab recently observed that resistance trained males significantly increased fat oxidation during exercise following five days of consuming a LCHF diet, suggesting that our participants may have "fat adapted". Regardless of what the explanation for a decreased RER during the $\dot{V}O_{2\ peak}$ test was, increasing fat oxidation while increasing performance is a favorable adaptation to HIIT.

Relative mean and peak and absolute mean power increased from pre-to post-intervention regardless of CHO timing. HIIT has previously been shown to increase relative peak and mean power (Astorino et al., 2012; Hazell et al., 2010) and absolute peak and mean power (Burgomaster et al., 2006; Burgomaster et al., 2008; Burgomaster et al., 2005; MacDougall et al., 1998; Whyte et al., 2010). Due to this study implementing a concurrent training model we cannot fully attribute increases in relative and absolute power output solely to HIIT as power output has also been shown to increase following RT (Balabinis et al., 2003; Kraemer et al., 1995; Leveritt, Abernethy, Barry, & Logan, 2003). Therefore, the most likely explanation is due to a combination of RT and HIIT. Increases in power output are most likely explained by increases in neuromuscular activation and strength of the leg musculature. We observed significant increases in 1-RM back squat and although not measured, participants may have increased neural drive, rate of force development, or had reductions in neuromuscular inhibition in the leg musculature (Aagaard et al., 2002; Aagaard et al., 2000). Evident by increases in relative and absolute power output, concurrent training resulted in favorable increases in power to body mass ratios.

A secondary aim of this investigation was to examine the effects of consuming a LCHF while completing a high-intensity training programs on biomarkers of health and performance. Resting blood glucose, testosterone, and insulin remained unchanged following the 4-week intervention. During 15 days of consuming a LCHF diet, our lab previously observed significant reductions in insulin following 5, 10, and 15 days of commencing the diet (Waldman et al., 2017). It may have been possible for insulin levels to initially decrease due to restricting CHO intake through the first 2 weeks, and then

levels increased back up to pre-intervention values after 4 weeks. Typically, investigations observing reductions in insulin are in unhealthy/sedentary adults (Volek et al., 2009). Significant reductions may also be due to the amount of CHO restriction. Volek et al. (2002) observed significant reductions in resting insulin levels in middle-aged healthy men following a 6-week ketogenic diet (~8% CHO). Reductions in insulin have been shown in other investigations examining similar populations and diets (Sharman et al., 2002; Urbain et al., 2017; Volek et al., 2001). Due to the pre-existing healthy levels of insulin and our LCHF diet not being as CHO restrictive as a ketogenic diet, our participants resting insulin levels remained unchanged.

Recently, Wilson et al. (2017) observed significant increases in testosterone levels following a 10-week RT program and ketogenic diet. This was one of the first studies to exam the influence of RT and ketogenic dieting on anabolic hormone status in humans. Independent of diet it is suggested that increasing daily fat intake increases lipid bioavailability (Wilson et al., 2017). This would in turn increase resting testosterone levels independent of RT, due to testosterone being a steroid hormone produced from cholesterol (Vingren et al., 2010). Furthermore, RT is a stimulus to increase resting testosterone levels. Previous investigations have found increases in resting testosterone levels following RT lasting 4 weeks (Staron et al., 1994), 6-8 weeks (Kraemer et al., 1998a), 6-10 weeks (Kraemer et al., 1999), 12 weeks (Moradi, 2015), and 14 weeks (Ahtiainen et al., 2003). With testosterone being an anabolic hormone responding to both RT and LCHF diets, we hypothesized significant increases in resting testosterone. However, the findings from this study suggests no differences in resting testosterone levels. Similar to our results with muscle thickness, our training program may have not

had enough training volume to induce changes in resting testosterone. Although Ahtiainen et al. (2003) observed increases in testosterone following a 7-week high-volume training cycle, a subsequent reduction in training volume during the final 7-week cycle led to a decrease in testosterone. Similar findings have also been observed in elite weight lifters (Hakkinen et al., 1987, 1988). With the variability of testosterone levels throughout a periodized training program and limited research of LCHF diets and RT, more research is needed to fully understand the interaction between diet induced and training induced increases in resting anabolic hormone concentrations. At this time, the specific increase in daily fat intake needed to elicit diet induced increases in testosterone is not fully understood. However, our data suggests that increasing daily fat intake to ~50% is not sufficient to increase testosterone even when accompanied with RT.

The tertiary aim of this investigation was to examine the impact of ingesting CHO and non-caloric flavored beverages on GID. To the authors knowledge this was one of the first investigations to examine the impact of CHO ingestion during RT and HIIT. Typically, investigations with GID are in endurance athletes with complaints of GID being induced from physiological, mechanical, or nutritional means (de Oliveira et al., 2014). Mechanical factors that can increase GID are related to body positioning and mechanical force produced during exercise. Running results in greater GID than cycling (Peters et al., 2000). Assuming that increased mechanical impact on the body increases GID, completing dynamic lower body resistance training could also increase GID. Exercise intensity also plays a role GID as increases in intensity decreases blood flow to the gastrointestinal organs and increases blood flow to the periphery (i.e. skin, working muscles) (Qamar & Read, 1987). Due to the effects of mechanical and physiological

factors leading to GI distress, it was hypothesized that participants would adapt to drinking CHO and non-CHO beverages during exercise and GID would decrease throughout the 4-week intervention.

Our statistical analysis of GID utilized a four-way RMANOVA to determine whether there were differences in symptoms of GID between HIIT and RT sessions across training sessions, weeks, and between groups. Ratings of nausea, reflux, and abdominal cramps were significantly higher post-exercise across all conditions when averaged between pre- and post-exercise. There were no differences between RT or HIIT sessions for any GID symptoms. This was surprising to the authors due to the different mechanical forces placed on the body during dynamic lower body RT and high-intensity cycling. Other investigations have observed increases in ratings of gas/flatulence for CHO containing beverages during endurance exercise (van Nieuwenhoven et al., 2005), but we observed no differences between beverages. This may be due to the 2-week familiarization period of drinking water we included. Two weeks may have been enough time to train the gastrointestinal tract and an addition of 30 g of CHO and a non-caloric placebo did not alter GID. Furthermore, when comparing overall ratings of GID with the same GID scale in runners (Wilson, 2017), our results suggests lower incidences in a population completing RT and HIIT.

Recently it has been suggested that the gut is an important organ and can be trained similar to skeletal muscle with the overall goal of reducing GID and increasing exogenous CHO availability (Jeukendrup, 2017). Assuming gastric emptying rates of CHO are maintained while consuming a LCHF diet (Castiglione, Read, & French, 2002), measuring GID during exercise could indirectly measure potential increases in gastric

emptying. However, reductions in stomach fullness were only observed across Weeks 1 and 2 with no other week changes and no differences between groups. With no changes in performance adaptations between the SUPP and NONSUPP groups, it is unlikely that meaningful changes in gastric emptying rates occurred to provide greater exogenous CHO availability for the SUPP group during exercise. Overall, our data suggests that flavored beverages cause significant increases in symptoms of GID. However, our GID results should be interpreted with caution as the scale used in the present investigation has not been externally validated during high-intensity exercise. Considering the popularity of ergogenic aids consumed during RT (i.e. amino acids, pre-workouts, CHO), more research in needed to further establish nutrient and exercise induced GID during high-intensity exercise and develop and validate GID scales.

There are several limitations to this investigation that need to be addressed. Due to the nature of the study, we were unable to blind the treatment groups to the participants or investigators. To truly examine the effects of CHO timing, the participants knew what their CHO timing schedule was. Although the investigators knew participants groupings, they remained unbiased throughout the study and provided verbal encouragement to each participant. We also provided participants in the NONSUPP group with non-caloric beverages consisting of the same fluid volume, flavor, and scent to ensure no placebo effect from not receiving anything.

A major limitation for all dietary studies is relying on self-reported dietary intakes. Our dietary adherence during the 4-week intervention was very good and we even recorded and reported dietary intakes twice a week along with randomly requesting dietary intakes weekly. Furthermore, participants were given several sample meals plans,

specific to where they typically ate meals (i.e. college meal plan options, self-cooking) to help them adhere to the diets and the principal investigator was available at all times to answer questions that arose during the study. However, without directly feeding participants we accept this as a limitation. We also told participants that they could not participate in any other form of structured exercise outside of our training sessions. If participants completed exercise outside of daily training session it could have altered daily energy expenditure and caused changes to fuel usage and storage. Finally, we had participants training at all times of the day. Training adaptations have been shown to change depending on training scheduling, but by requiring participants to train at the same time each day we hoped to minimize diurnal variations potentially causing time-dependent training effects (Chtourou & Souissi, 2012).

Although there were several limitations to our investigation, there are were also several strengths. First, as mentioned earlier, we provided supervised training during each training session. Supervised training has been shown to provide superior training adaptations compared to unsupervised training (Mazzetti et al., 2000). A limitation of other CHO timing studies are the differences in PRO intakes between experimental groups (Aragon & Schoenfeld, 2013). We matched groups for PRO, fat, and CHO intake with the only difference being the timing of CHO. Both groups also ingested 25 g of PRO one hour prior to and 25 g immediately following each exercise session to ensure adequate PRO levels to promote maintenance of positive protein balance. By matching nutrient intakes between treatment groups, the sole difference in the results of the investigation were based on timing of 70 g of CHO.

CHAPTER VI

CONCLUSION

The purpose of this investigation was to examine the impact of CHO timing when consuming a LCHF diet and participating in a 4-week high-intensity training program. Our results suggest that (1) concurrently RT and HIIT while consuming a LCHF significantly increases muscular strength, $\dot{V}O_{2\ peak}$, and power output, (2) does not improve resting biomarkers of health and performance, and (3) does not provide favorable body composition changes (specifically fat mass). Furthermore, consuming a LCHF diet and completing HIIT eight times resulted in a significantly lower exercise RER, suggestion participants "fat adapted" and/or increased reliance of fat during aerobic exercise due to favorable training adaptions and increased TTE. However, there were no differences between the SUPP and NONSUPP groups for any performance outcome.

Ingesting CHO and non-caloric flavored beverages resulted in similar GID during RT and HIIT. Participants GID was lower than reported GID in endurance athletes (Wilson, 2017), but there were no meaningful subjectively measured adaptions across weeks. Chronically ingesting flavored beverages during high-intensity exercise may be an important strategy for non-endurance athletes who predominately RT and consuming nutrition during training sessions to minimize GID. Since this was one of the first investigations examining GID in non-endurance exercise, it was an important step to examine GID that could potentially cause performance decrements. More research with

resistance trained populations is needed to further examine the potential impact between GID and performance.

Overall, it may be suggested that daily CHO intake and timing of CHO intake is not as important as current guidelines suggest (Kreider et al., 2010; Slater & Phillips, 2011). Future researchers can use these results to closely examine daily CHO needs for individuals who predominately complete high-intensity exercise. In agreement with other LCHF diet research, reducing CHO intake does not result in strength decrements (Gregory et al., 2017; Kephart et al., 2018; Van Zant et al., 2002) and significant increases in strength and endurance can be made (Wilson et al., 2017). Finally, it should be acknowledged that we used a sample of recreationally trained college aged males and our results may not translate to athletic populations. However, future studies should target athletic populations (i.e. body builders, weightlifters, anaerobic athletes), lower CHO diets, and longer duration studies to further examine the importance of CHO timing.

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APPENDIX A

IRB APPROVAL

Subject: Approval Notice for Study # IRB-17-461, The Importance of Carbohydrate Timing During High-Intensity Training while Consuming a Low Carbohydrate Diet

Date: Wednesday, September 6, 2017 at 8:57:49 AM Central Daylight Time

From: jbr6@msstate.edu

To: jws597@msstate.edu, ajt188@msstate.edu, bds243@msstate.edu, bjf2@msstate.edu, bmk216@msstate.edu, bn241@msstate.edu, cla297@msstate.edu, fgp13@msstate.edu, hbs193@msstate.edu, hsw78@msstate.edu, jcs652@msstate.edu, jdb1089@msstate.edu, jgl5@msstate.edu, jmd589@msstate.edu, mjm639@msstate.edu, plw135@msstate.edu, sab480@msstate.edu, st1527@msstate.edu, ymb14@msstate.edu

Protocol ID: IRB-17-461
Principal Investigator: JohnEric Smith
Protocol Title: The Importance of Carbohydrate Timing During High-Intensity Training while Consuming a Low Carbohydrate Diet
Review Type: FULLBOARD
Approval Date: September 06, 2017
Expiration Date:August 15, 2018

The above referenced study has been approved. To access your approval documents, log into myProtocol and click on the protocol number to open the approved study. Your official approval letter can be found under the Event History section. For non-exempt approved studies, all stamped documents (e.g., consent, recruitment) can be found in the Attachment section and are labeled accordingly.

If you have any questions that the HRPP can assist you in answering, please do not hesitate to contact us at irb@research.msstate.edu or 662.325.3994.

APPENDIX B

INFORMED CONSENT

Mississippi State University
Informed Consent Form for Participation in Research

Title of Research Study: The Importance of Carbohydrate Timing During High-Intensity Training while Consuming a Low Carbohydrate Diet.

Study Site: McCarthy Gymnasium, J.A. Chromiak Applied Physiology Lab, Spin Room Joe Frank Sanderson Center

Researchers: Ben Krings, JohnEric Smith, Matt McAllister, Brent Fountain, John Lamberth, Hunter Waldman, Brandon Shepherd, Steven Basham, Saira Talwar, Peyton Williamson, Jonathan Barber, Jon Swain, Alana Turner, Ffion Price, John Dodd, Cecelia Andreo

Purpose

The purpose of this study is to examine the effects of timing of daily carbohydrate ingestion during a short term-high intensity exercise program while consuming a low carbohydrate, high fat diet on exercise performance, blood markers, body composition, and fuel mobilization and utilization during exercise.

Target Recruitment

Recruiting procedures are targeted toward individuals who meet the inclusion/exclusion criteria as listed below. You may be asked to suggest names and contact information for others you know that may be interested in and qualified to participate. Recruitment flyers will be displayed all around Mississippi State's Campus and we will send an email to undergrad kinesiology students to advertise the study once consent from the department head is given.

Inclusion/Exclusion Criteria

Subjects must meet the intermediate resistance training experience classification according to the National Strength and Conditioning Association. To meet this classification, subjects must have completed resistance training at least 3 days per week for the past 2-6 months. Subjects must also meet the minimum physical activity guidelines according to the American College of Sports Medicine, completing at minimum 150 minutes of moderate, 75 minutes of vigorous, or a combination of moderate and vigorous physical activity per week. Subjects must refrain from taking caffeinated supplements (thermogenics, pre-workouts, etc.), but are allowed traditional sodas and coffee, for the duration of the two-week familiarization period and four-week nutrition/dietary intervention period.

Subjects will be excluded if they report taking banned substances (steroids, etc.) and miss more than two training sessions during the four-week nutrition/training intervention period. This could be two resistance training

sessions, two high-intensity interval training (HIIT) sessions, or a combination of one of each. They may also be excluded from participating if they check yes to any health conditions on the PAR-Q form.

Additionally, the researchers reserve the right to terminate both exercise and your participation in the study if proper execution of each movement cannot be safely performed throughout the duration of a resistance training session. This includes but not limited to: an inability to maintain a neutral back, upright head, controlled and coordinated execution of each lift, and proper breathing mechanisms that help ensure safety for both lifters and spotters.

Finally, after a review of the three-day food log, if the percentage for dietary fat is greater than 40% of their total daily caloric intake, you will be excluded from the study. If the subjects do not adhere to the dietary guidelines of the study, the researchers reserve the right to terminate completion of the study.

Brief Overview of Study

Subjects will complete a total of 39 sessions requiring ~34 hours of time commitment. The study is broken down into five periods: 1. pre-testing, 2. familiarization training, 3. mid-testing, 4. four-week training/nutrition intervention, and, 5. post-testing (Figure 1). During the pre-testing period, subjects will be informed of the study details and consent if they agree to participate. After agreeing to participate subjects will complete a lifestyle and health questionnaire and a three-day dietary recall. They will also have height, weight, a Wingate anaerobic test, peak oxygen consumption, and upper and lower body strength assessed. Upon competition of pre-testing, subjects will commence a two-week familiarization period consisting of six resistance training sessions and one HITT session. After familiarization, the mid-test period consists of a peak oxygen consumption test, a resting blood sample, exercise blood samples, and body composition assessments (via BodPod and ultrasound). During the final session of the mid-test period, subjects will be made aware of the experimental group they are assigned (SUPP or NON-SUPP) and be given dietary guidelines and resources to help them adhere to the diet. Leading up to this point, subjects will be on their habitual diet. Next, the four-week nutrition/training intervention begins and includes a total of 20 sessions (12 resistance training and 8 HIIT). Monday, Wednesday, and Friday are resistance training and Tuesday and Thursday are HIIT days. Along with training, subjects will be consuming a low carbohydrate, high fat diet (~20% carbohydrate and protein, ~60% fat) and their caloric intake will be based on body composition and physical activity level. They will also have a specific daily nutrition timing schedule, specifically with their carbohydrate intake, depending upon their experimental group assigning. After four weeks of the training/nutrition intervention post-testing will begin. Post-testing includes a resting blood sample, peak oxygen consumption testing, exercise blood samples, body composition assessment (via BodPod and ultrasound), and upper and lower body strength assessments.

Details of Each Session

Pre-Testing

1. **Session 1-** This session will serve as a baseline meeting. During this time, you will be informed of the potential risk and details of the study. If you consent to participating in the study, a physical activity readiness form, lifestyle questionnaire, and general health questionnaires will be completed. The PAR-Q will be used to ensure that you are considered low risk for a cardiovascular and musculoskeletal problem. You will be asked to complete a three-day dietary recall for the three days (two weekdays and one weekend day) following your baseline session using myfitnesspal phone app. You will be shown how to use myfitnesspal during this session. A handout describing the proper lifting techniques of the complex resistance exercises will also be handed out.
    a. Time Commitment and Location- 60 minutes @ McCarthy Gym Applied Physiology Laboratory

2. **Session 2-** During this session you will have height, weight and a Wingate anaerobic test assessed. The Wingate test will use a cycle ergometer and require 30 seconds of maximal effort.
    a. Time Commitment and Location- 30 minutes @ McCarthy Gym Applied Physiology Laboratory

3. **Session 3-** Peak oxygen consumption will be assessed using a metabolic cart and cycle ergometer, during a cycling protocol slowly increasing intensity until volitional fatigue. This test will serve as a familiarization to the protocol and equipment. Since this test serves as a familiarization, there is no collection of blood.
    a. Time Commitment and Location- 30 minutes @ McCarthy Gym Applied Physiology Laboratory

4. **Session 4-** Upper body strength will be collected on bench press, shoulder press, wide grip row, upright row, and hammer curls.
    a. Time Commitment and Location- 60 minutes @ McCarthy Gym Applied Physiology Laboratory

5. **Session 5-** At least two days after session four, you will complete similar strength testing, but with back squat, Romanian deadlift, step-ups, Bulgarian split squats, and lunges.
    a. Time Commitment and Location- 60 minutes @ McCarthy Gym Applied Physiology Laboratory

Familiarization

(**Sessions 6-12)-** Before you begin the four-week high intensity exercise/dietary intervention, there will be a two-week familiarization period. This period will serve

as time to become comfortable with the fluid ingestion schedule and the exercises. You will train for a total of one hour on Monday, Wednesday, and Friday for two weeks (6 total sessions) @ McCarthy Gym Applied Physiology Laboratory (six-hour time commitment). Monday will be lower body resistance training, Wednesday will be upper body resistance training, and Friday will be full body resistance training (Table 1). You will also be required to attend one cycling HIIT session. A total of four classes will be offered. (1 session) @ Spin Room in the Joe Frank Sanderson Center (30-minute time commitment)

Mid-Testing

1. **Session 13-** Blood will be collected following at least a 10-hour fast via butterfly stick. A total of 14 mL of blood will be collected into a sealed vacutainer. Following the fasting blood draw, peak oxygen consumption will be assessed using a metabolic cart and cycle ergometer, during a 20-minute cycling protocol slowly increasing intensity until volitional fatigue. This test will serve as a pre-test before beginning the four-week training/nutrition intervention. During this test, a total of six finger stick blood measures will be taken. Your finger will be pricked using a lancet and 0.3 μL collected six times for a total of 1.8 μL.
    a. Time Commitment and Location- 45 minutes @ McCarthy Gym Applied Physiology Laboratory

2. **Session 14-** The purpose of this session will be to assess body composition via BodPod and ultrasound. This session will take place following at least four hours of not eating. You will be instructed to arrive wearing compression shorts. Ultrasound will be measured on six muscles; biceps, triceps, chest, calf, hamstrings, quadriceps.
    a. Time Commitment and Location- 45 minutes @ McCarthy Gym Applied Physiology Laboratory

3. **Session 15-** During this session, you will be informed of the experimental group you are in and be given nutritional information regarding your intervention group. You may ask any necessary questions regarding the dietary intervention. Sample meal plans will be provided.
    a. Time Commitment and Location- 30 minutes @ McCarthy Gym Applied Physiology Laboratory

High-Intensity Exercise Training/Nutrition Intervention

(**Sessions 16-35)-** Over the course of the next four weeks there will be a total of 20 training sessions, three resistance training (Table 1) and two cycling HIIT sessions (Table 2) per week. Each resistance training session will last ~60 minutes and HIIT session will last ~45 minutes for a total of ~18 hours time commitment. All resistance training sessions will take place in McCarthy Gym Applied Physiology Laboratory and HIIT in the Spin Room in the Joe Frank

Sanderson Center. During each Friday resistance training workout, you will have blood glucose collected via fingerstick procedure immediately before exercise, during the middle of exercise, and at the end of exercise. Your finger will be pricked using a lancet and a 0.6 μL blood sample collected three times for a total of 1.8 μL. During each training session (resistance training and HIIT), depending on the group which you are assigned, you will follow the following supplementation schedule: Both groups will ingest one scoop of protein powder (25 grams) on their own, which will be provided to you before beginning the four-week nutrition/training intervention, one hour prior to coming in to each training sessions. No other intake of food containing any caloric value is allowed within the one hour period before exercising. Immediately upon arriving at the training session you will receive a shaker bottle with either 30 grams of carbohydrates or an artificially flavored non-caloric beverage in 500 mL of water. You will be advised drink 100 mL of the beverage after the warmup and after the final set of each of the first four exercises for resistance training sessions. 100 mL will be ingested immediately before and after the warmup, and after each of the first three sprints during HIIT days. Immediately following the training sessions, you will receive a beverage containing 40 grams of carbohydrate and 25 grams of protein (SUPP group) or a beverage containing only 25 grams of protein (NON SUPP group). For the one hour after exercise, you will not be allowed to ingest any other nutrition source like the pre-one hour exercise guidelines. During the four-week nutrition/training intervention, you will be asked to track your daily nutrition intake on myfitness pal. You will also be asked to provide screen shots of your myfitnesspal to the principal investigator on Tuesdays and Fridays (corresponding to food intake from Mondays and Thursdays). You will be randomly asked to provide screen shots of your food intake throughout the study to ensure compliance.

Post-Testing

1. **Session 36-** The purpose of this session will be to assess body composition via BodPod and ultrasound. This session will take place following at least four hours of not eating. You will be instructed to arrive wearing compression shorts. Ultrasound will be measured on six muscles; biceps, triceps, chest, calf, hamstrings, quadriceps. Following body composition assessments, a Wingate anaerobic test will be completed on cycle ergometer and require 30 seconds of maximal effort.
    a. Time Commitment and Location- 60 minutes @ McCarthy Gym Applied Physiology Laboratory

2. **Session 37-** Blood will be collected following at least a 10-hour fast via butterfly stick. A total of 14 mL of blood will be collected into a sealed vacutainer. Following the fasting blood draw, peak oxygen consumption will be assessed using a metabolic cart and cycle ergometer, during a 20-minute cycling protocol slowly increasing intensity until volitional fatigue. This test will serve as a post-test to the four-week training/nutrition intervention. During this

test, a total of six finger stick blood measures will be taken. Your finger will be pricked using a lancet and 0.3 μL collected six times for a total of 1.8 μL.
   a. Time Commitment and Location- 45 minutes @ McCarthy Gym Applied Physiology Laboratory

6. **Session 38-** Upper body strength will be collected on bench press only.
   a. Time Commitment and Location- 30 minutes @ McCarthy Gym Applied Physiology Laboratory

7. **Session 39-** At least two days after session 38, lower body strength testing will be collected on back squat only.
   a. Time Commitment and Location- 30 minutes @ McCarthy Gym Applied Physiology Laboratory

Experimental Procedures

Dietary Intervention/Carbohydrate Timing Schedule

Over the course of the first 14 sessions you will maintain your habitual diet. During session 15, you will be informed of which group you are in and provided with a set of guidelines for consuming a low carb, high fat diet. This procedure will be overseen by a registered dietician that Mississippi State has on campus. This diet will consist of eating a macronutrient ratio ~60% total energy from fat and ~20% energy from protein and carbohydrates, respectively. These macronutrient percentages will be based off your estimated total caloric intake, (calculated from your body composition results and a physical activity factor of active, session 14).

Involvement in this study will require you to keep a daily food journal/log through myfitness pal, an online food database that makes tracking macronutrient percentages relatively easy. Familiarization with myfitness pal will take place during session 1. You will also be provided a survey mid-way through the study so that your energy, mood, mental fatigue, etc. can be monitored and (calories) adjusted if need be. If you begin to see significant fluctuations ~5 lbs within a week, we will reassess your caloric intake and make necessary adjustments.

You will be randomly assigned to the SUPP group or NON-SUPP group. Both groups will ingest the same daily diet, listed above, with the only difference being the timing of when you ingest your carbohydrates. The SUPP group will ingested 30 grams of provided carbohydrates during and 40 grams of provided carbohydrates immediately after exercise. 30 grams of carbohydrates (in 500 mL of water) will be administered during each resistance training and HIIT sessions during the four-week intervention period. Immediately after exercise, if you are in the SUPP group, you will receive 40 grams of carbohydrates and 25 grams of protein. The NON-SUPP group will receive the same volume of fluid during exercise, but with an artificially flavored non-caloric beverage. After exercise, you will receive 25 grams of protein, but no carbohydrates. One hour before you are

scheduled to train, you will be asked to ingest 25 grams of protein powder, and nothing else, besides water, until your training session. You will be provided with the protein powder so you can take it on your own time and don't have to come to the lab and spin room. After training you will ingest your post-exercise beverage and nothing else, besides water, until one-hour post exercise. If you are in the SUPP group you will ingest your remaining carbohydrate evenly distributed throughout the day (The investigators will inform you of how many carbohydrates you have left to consume for the rest of the day). If you are in the NON-SUPP group you received no carbohydrates during exercise, so you get 2g/kg of carbohydrate outside of training and will be instructed to distribute carbohydrates evenly throughout the day. During the weekends, when you are not training, you are still requested to follow the daily dietary intake guidelines.

Blood Draws/Fingersticks

Blood samples will be collected a total of 6 times (Sessions 13, 20, 25, 30, 35, 37). Two resting blood samples will be collected (Sessions 13 and 37) via antecubital venipuncture with a butterfly needle. A total of 14 mL per session will be collected into a sealed vacutainer. Prior to drawing blood via venipuncture, the site will be dry cleaned with sterile gauze then sterilized with an alcohol wipe. The site will then air dry and a butterfly needle will be inserted into the antecubital vein. Upon collecting 14mL of blood, the needle will be removed and a sterile gauze pad will be place on the site with pressure applied. Pressure will be applied for ~2 minutes to ensure that bleeding has stopped and a bandage will be applied.

The remaining blood samples will be collected via capillary fingerstick. During sessions 13 and 37 a total of 6 fingersticks (0.3 μL each stick= 1.8 μL total) will be administered with a lancet and collected for analysis of blood lactate each session. During sessions 20, 25, 30, and 35 a total of 3 fingersticks (0.6 μL each stick= 1.8 μL) will be administered with a lancet and collected for analysis of blood glucose during each session. During fingerstick blood collections, the pointer, middle, and ring fingers of the right hand may be pricked. The middle finger will be used, preferably, and a site on the lateral side near the fingernail end of the finger will be pricked. The site will be wiped with sterile gauze and then sterilized with an alcohol wipe. After being air dried, the site will be pricked using a self-retracting lancet. The first drop of blood will be wiped away using sterile gauze and the second drop will be collected on to a lactate strip. After collecting a very small amount of blood, you will be given a piece of sterile gauze and instructed to squeeze your finger into your palm. This process will maintain pressure to stop the bleeding. On the subsequent blood draws, during the peak oxygen consumption testing, the same site may be used. The researcher will squeeze the end of your finger to collect blood. If the same site bleeds again, instead of using a lancet to prick another site, the same site will be used. This process is used to minimize the amount of fingersticks. However, due to the variability in individuals bleeding, there is a potential for 6 fingersticks during the peak oxygen consumption testing. The order of fingerstick sites will go as follows

if necessary, lateral sides of middle finger, lateral sides of ring finger, and lateral sides of pointer finger. When a finger is done being used for blood collection and pressure has been applied to stop the bleeding a bandage will be applied.

All antecubital venipunctures and fingersticks will be completed in the Applied Physiology Laboratory by university approved individuals.

Upon analysis of blood data, which will occur following the end of the study, there may be blood values that we read as being considered abnormal. If this is the case, we will contact you. To follow this up, you may want to consider a follow-up with your primary care physician.

Resistance Training

Pre-and post-resistance training familiarization and training/nutrition intervention, true one repetition maximum (1RM) (bench press and back squat) and predicted 1RM (shoulder press, upright row, wide grip row, hammer curls, Romanian deadlift, Bulgarian split squat, step-ups, and lunges) testing will take place. During true 1RM testing, subjects will be instructed to lift a light resistance for 5-10 repetitions followed by a 1-minute rest. Depending on the exercise, 4.5-18 kilograms (10-40 pounds) will be added to the barbell and 3-5 repetitions will be completed, followed by 2 minutes of rest. 4.5-18 kilograms (10-40 pounds) will be added again to the bar and 3-5 repetitions will be completed, followed by 2 minutes of rest. After this final warmup, subjects will try to hit a 1RM within 3-5 sets. During predicted 1RM testing, subjects will try and lift within a target range of 2-5 maximum repetitions to predict 1RM. A three-set approach will be taken (one warm-up set with light weight (5-10 repetitions), 1 warmup 50-80% perceived 1-RM (3-5 repetitions), and 1 max estimation set).

The resistance training program is designed using a linear periodization model starting with higher volume/lower intensity and increasing to lower volume/higher intensity. Based upon the true and predicted 1RM testing subjects will have an individualized plan. Exercises that did not require 1RM testing (i.e., triceps dips, hip abductors, Russian twists, etc.) will use body weight, elastic bands, and medicine balls. Each subject will pick a time that fits most convenient for their schedule to train and keep that time for every resistance training session. Subjects will be train in at maximum groups of three with other subjects. Each session will be supervised by a Certified Strength and Conditioning Specialist (NSCA) to ensure proper technique, adherence to exercise, and adherence to the supplementation schedule. Prior to each training resistance training session, subjects will have their body weight measured and recorded. On Monday's and Wednesday's each exercise has a set amount of repetitions. Subjects will be instructed to achieve the repetition count and if not, achieve as near to the count as they can. On Friday's, there will be four exercises used to measure weekly performance. The second set of back squat and bench press and third set of pushups and pullups will be completed for as many repetitions as possible and recorded. The rest times for between each exercise is listed below Table 1.

On Monday's only during the nutrition/training intervention four-week period, subjects will complete a gastrointestinal (GI) scale pre-and post-exercise. This will be used to assess GI distress. The GI scale uses a 0-10 Likert scale with ordinal scoring and scores corresponding to 0 (no discomfort), 5 (moderate discomfort), and 10 (unbearable discomfort). This scale will be used to assess both lower and upper GI tract issues including, nausea, regurgitation/reflux, stomach fullness, abdominal cramps, flatulence/gas, and urge to defecate.

High-Intensity Interval Training Sessions

All HIIT sessions will take place in the Spin Room located in the Joe Frank Sanderson Center, meaning the subject must be a full-time student to have access to the facility. Each Tuesday and Thursday, two sessions will be provided, one in the mid-morning and one mid-afternoon. Subjects will be allowed to pick a time and stay in that time slot for all eight experimental sessions. Upon arrival to the Spin Room, subjects will be fitted with a heart rate monitor. During the HIIT protocol, subjects will be instructed to pedal as fast as they can and achieve an effort at 90% of their heart rate max. If the subject does not reach this value, they will be instructed to increase the resistance or pedal faster. Heart rate will be continuously measured on an iPad by an investigator. Subjects will train in groups of no more than 15 during one session. At least three investigators will be at each HIIT session. On Tuesday's only during the nutrition/training intervention four-week period, subjects will complete a gastrointestinal (GI) scale pre-and post-exercise, as described above.

Wingate Anaerobic Test

Wingate testing will take place on a cycle ergometer. Subjects will begin with a five-min warm-up against a self-selected pace. Following the warm-up, the Wingate test will begin. Ten seconds prior to beginning the test all the resistance will be removed from the cycle ergometer and at six seconds prior to the test, participants will be instructed to maximally increase their cadence. A resistance of 0.075 $kg \cdot kg^{-1}$ body mass of resistance will be magnetically added. Participants will be instructed to cycle with maximal effort for 30 seconds while keeping their buttock on the seat. Verbal encouragement will be provided by the investigators. Following completion of the test, at least a 5-minute cooldown will be completed on a cycle ergometer.

Peak Oxygen Consumption Test

The $\dot{V}O_{2peak}$ test will be conducted on a cycle ergometer. The first test will not require a ten hour fast and the final two will require at least a ten hour fast. Participants will be fitted with a heart rate monitor and wear a snorkel like apparatus to measure gas exchange and breathing. The test begins by cycling at 100 watts for three minutes. Following the first stage, the next three stage will last three-minutes each and correspond to 150, 200, and 250 watts. Each

subsequent stage will increase by 25 watts each minute. The test will stop at volitional fatigue. During the test, subjective fatigue will be measured using the 0-10 modified Borg rating of perceived exertion scale.

Body Composition

Body composition will be assessed using a BodPod and ultrasound. The BodPod measures body composition (% body fat, fat mass, and fat free mass) via air displacement plethysmography. Subjects will arrive following at least a four hour after eating and be advised to defecate or urinate if need be and strip down to compression shorts. They will then be provided a swim cap and enter the BodPod for five minutes. After exiting the BodPod, subjects will have cross section area of the biceps, triceps, chest, hamstrings, quadriceps, and calf muscles measured using an ultrasound. A wand with lubricant gel will be gently rubbed on the target muscle twice for each muscle.

Figure 1.

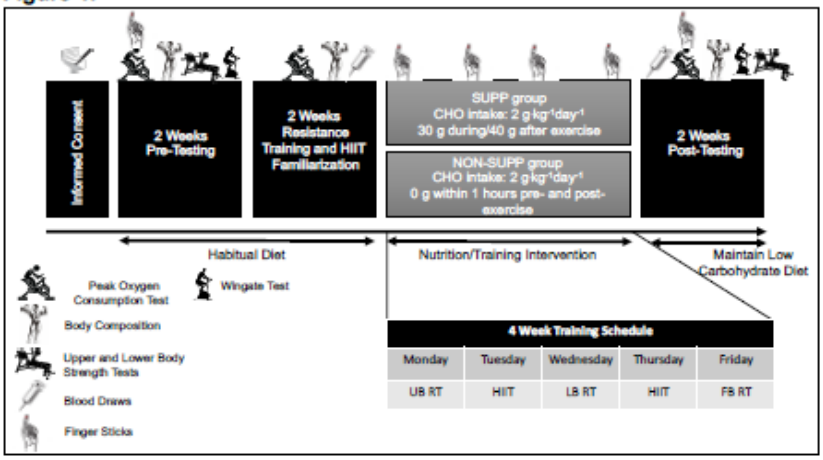

Table 1.

Monday Lower Body			
Exercise	Sets	Repetitions	Intensity
Back Squat†	3	12/8/6	67/80/85%
Romanian Deadlift†	3	12/8/6	67/80/85%
Step-Ups†	3	12/10/8	67/75/80%
Bulgarian Split Squats†	3	12/10/8	67/75/80%
Calf Raises^	3	12/15/20	Body Weight
(1) Hip Abduction^	3	12/10/8	Bands
(2) Hip Adduction ^	3	12/10/8	Bands
Curl-ups*	2	20/25/30	Body Weight
Russian Twists*	2	20/25/30	Medicine Ball
Wednesday Upper Body			
Exercise	Sets	Reps	Intensity
Barbell Bench Press†	3	12/8/6	67/80/85%
Landmine Wide Grip Row†	3	12/8/6	67/80/85%
Shoulder Press†	3	12/8/6	67/80/85%
Upright Row†	3	12/8/6	67/80/85%
(1) Hammer Curls^	3	12/8/6	67/80/85%
(2) Triceps Dips^	3	10/15/20	Body Weight
Curl-ups*	2	20/25/30	Body Weight
Planks*	2	45/60/75 secs	Body Weight
Friday Full Body			
Exercise	Sets	Reps	Intensity
Back Squat†	2/ final to failure	12/8/6	67/80/85%
Bench Press†	2/ final to failure	12/8/6	67/80/85%
Lunges†	3	12/10/8	67/75/80%
Shoulder Press†	3	12/8/6	67/80/85%
(1) Pushups^	3/ final to failure	10/15/20	Body Weight
(2) Pullups^	3/ final to failure	6/8/12	Body Weight
Planks*	2	45/60/75 secs	Body Weight
Side Planks*	2	30/45/60 secs	Body Weight

Exercises listed as (1) and (2) will be completed as a superset; *30 seconds rest between sets, ^60 seconds rest between sets, †120 seconds rest between sets. Hyphens between repetitions and intensity represent what is to be completed the first two weeks, weeks three and four, and weeks five and six. Half of the repetitions will be completed on each leg for the unilateral exercises (step-ups, Bulgarian split squats, lunges)

Table 2.

	Week 1	Week 2	Week 3	Week 4
Days per week	2 days	2 days	2 days	2 days
Volume	30 s x 4 sprints	30 s x 4 sprints	30 s x 5 sprints	30 s x 6 sprints
Rest Periods	4 minutes	4 minutes	4 minutes	4 minutes
Intensity	"All out" effort	"All out" effort	"All out" effort	"All out" effort
Heart Rate	~90 % HR max	~90 % HR max	~90 % HR max	~90 % HR max
Warm-up	5 minutes	5 minutes	5 minutes	5 minutes
Cool-down	10 minutes	10 minutes	10 minutes	10 minutes
Time per session	29 minutes	29 minutes	33.5 minutes	38 minutes
Time per week	58 minutes	58 minutes	67 minutes	76 minutes

Risks or Discomforts

Completion of resistance training and HIIT may lead to muscle tears or soreness, substantial fatigue, dizziness, headache, acute elevation of blood pressure, headache, acute elevation of blood pressure, heart attack, stroke, rhabdomyolysis, or sudden death. According to the American College of Sports Medicine, the absolute risk of sudden cardiac death during vigorous physical activity has been estimated at one per year for every 15,000 – 18,000 people. (Siscovick, D.S., Weiss, N.S., Fletcher, R.H., & Lasky, T. (1984). The incidence of primary cardiac arrest during vigorous exercise. N Engl J Med, 311(14), 874-877.) Individuals trained in CPR, AED, and First Aid will be present during training sessions, and the training will be terminated if complications occur.

Possible complications can arise with having blood drawn from both the antecubital space and finger such as: allergies, inflammation, fainting, dizziness, bruising at the puncture site (hematoma), infection, nerve injury, and arterial puncture or laceration. If you have ever experienced any of these complications in the past, bring them to the attention of the researcher every time you have your blood drawn. The researchers may use nitrile and/or latex exam gloves when performing venipunctures and fingersticks. If you have any allergies to these materials let the researchers know and they can use latex-free and nitrile-free exam gloves. Alcohol wipes will be used to prepare the blood draw/fingerstick sites and if you have any allergies to alcohol wipes please let the researchers know.

The risks associated with consuming a low carbohydrate diet include: stomach stress, mood swings, foggy cognition, and altered lipid profiles. If you feel any of these risks please contact the researcher.

Finally, if you are not used to drinking fluid during exercise, both carbohydrate based and artificially flavored, you may feel stomach distress. If

this occurs, please let the researcher know.

Compensation

There is no direct compensation for the participants outside of receiving nutritional supplements for the study.

Benefits

Since each session is complete with the instruction of an investigator, you will receive supervised and guided exercise training. Additionally, you will have several metabolic tests and blood samples collected which may provide you with health and performance related data to further improve your training after completing the study.

Confidentiality

Your participation in this study is voluntary. You can withdraw or refuse to participate or ask any questions at any time without consequences. You will be assigned a subject number that will allow us to remove your personal identifying information from data. Due to the nature of group training utilized in this study, you will train with other subjects, but any information of subjects participation in the study will not be used outside of the experimental sessions. Although subjects are completing sessions with other subjects, the investigators will maintain privacy of each individual subjects collected data. Your data, including data sheets and electronic data, will only be identified by your participant number during the study. The participant's identity will be kept in a separate data file after each subject's completion of all data collection sessions in the study. The key code will be destroyed no later than December 31, 2017. Paperwork with identifying information (signature) will be paper shredded after collection (session 1) and ensuring the inclusion criteria is met. However, electronic data files will be kept indefinitely, but have no identifying information.

All hard copy data (informed consents) will be kept in a locked file cabinet. Only Dr. JohnEric Smith and Ben Krings will have access to the room and informed consents. If the data are reported at a scientific meeting or published in a scientific journal, only the group data will be reported. The data collection process will be completed by December 2017. The researchers reserve the right to terminate testing if there are potential hazards to subject's safety or wellbeing. Please note that these records will be held by state entity and therefore are subject to disclosure if required by law. Research information may be shared with the MSU Institutional Review Board (IRB) and the Office for Human Research Protections (OHRP) and others who are responsible for ensuring compliance with laws and regulations related to research.

Questions

If you have any questions about this research project, please feel free to contact Ben Krings (715-279-1372) and JohnEric Smith (941-592-5575).

For questions regarding your rights as a research participant, or to discuss problems, express concerns or complaints, request information, or offer input, please feel free to contact the MSU Research Compliance Office by phone at 662-325-3994, by e-mail at irb@research.msstate.edu, or on the web at http://orc.msstate.edu/humansubjects/participant/.

Research-related injuries

The risk associated with resistance training include the risks of sprains, strains, tears, muscle soreness, rhabdomyolysis, and fatigue.

MSU will not provide any payment to you or for your treatment if you are harmed as a result of taking part in this study.

In addition to reporting an injury to Ben Krings at 715-279-1372 and to the Research Compliance Office at 662-325-3994, you may be able to obtain limited compensation from the State of Mississippi if the injury was caused by the negligent act of a state employee where the damage is a result of an act for which payment may be made under §11-46-1, et seq. Mississippi Code Annotated 1972. To obtain a claim form, contact the University Police Department at *MSU UNIVERSITY POLICE DEPARTMENT, Williams Building, Mississippi State, MS 39762, (662) 325-2121.*

Voluntary Participation

Please understand that your **participation is voluntary.** Your **refusal to participate will involve no penalty or loss** of benefits to which you are otherwise entitled. You **may discontinue your participation** at any time without penalty or loss of benefits.

Options for Participation
Please initial your choice for the options below:

___The researchers may contact me again to participate in future research activities.

___The researchers may NOT contact me again regarding future research.